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ARCHIVES OF OTOLARYNGOLOGY

VOLUME 17 JANUARY, 1933 NUMBER 1

EFFECT OF SILVER NITRATE ON THE NASAL MUCOSA OF RABBITS

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In the treatment of vasomotor rhinitis and other conditions characterized by hyperesthesia of the nasal mucous membranes, good clinical results are usually obtained by the injection of alcohol into the sphenopalatine ganglion and by the application of solutions of silver nitrate to the mucous membrane over this ganglion. Following the latter method of treatment, beneficial results may appear in from one to two weeks and last for a year.

In an attempt to determine why such benefit should result from local applications of silver nitrate, series of experiments were performed on rabbits, 20 per cent solution of silver nitrate being applied to the nasal mucosa. At varying intervals afterward, the animals were killed, and sections of the nasal mucous membrane, which had been treated, were studied histologically.

EXPERIMENTAL DATA

Rabbits, apparently, do not have a true sphenopalatine ganglion as does man. The nerve supply to the mucosa of the posterior portion of the nose, the ethmoidal and antral recesses, the turbinate bones and the hard and soft palate is derived from the second division of the fifth cranial nerve. The second division of the fifth nerve passes beneath and mesially to the eye through the superior maxilla, and its terminal branches are distributed to the skin and soft tissues of the upper jaw and cheeks. Posteriorly in its course, the nerve supply to the posterior part of the palate is given off, and a large anterior palatine branch passes forward close to the floor of the nose to be distributed to the soft tissues of the anterior half of the palate. The posterior palatine branches, usually three, emerge through separate foramina in the lateral bony nasal wall, posterior to the narrow bony palate, to be distributed to the lining of the hard and soft palate. Anterior to these, a larger branch is given off. It enters the nasal cavity through a foramen situated in the lateral

From the Division of Experimental Surgery and Pathology and the Section on Otolaryngology and Rhinology, the Mayo Clinic.

bony nasal wall near its posterior extremity, just below the posterior or ethmoidal turbinate mass and a few millimeters above the posterior margin of the hard palate. This branch and its subdivisions are distributed to the lining of the lateral nasal wall, the turbinate masses and the subjacent nasal recesses; this was the nerve treated in the experiments.

An ordinary long intravenous needle with a blunted point and containing a cannula was introduced into the nostril and passed backward close to the septum and to the floor of the nose to avoid contact with the turbinate masses. At a depth of 4 cm. in rabbits weighing 1 Kg. and at a depth of from 4.7 to 5.7 cm. in rabbits weighing from 2.5 to 3.5 Kg. the opening of the foramen was encountered by

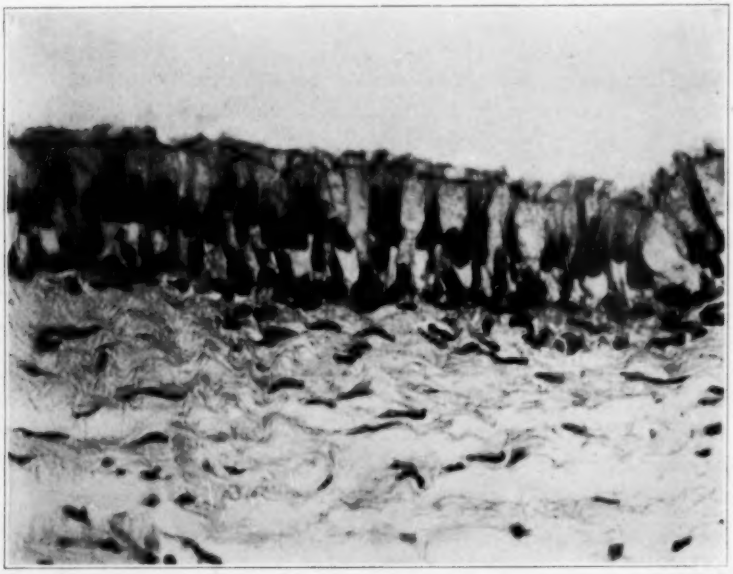

Fig. 1.—Normal nasal mucous membrane of a rabbit. Ciliated columnar epithelium and loose areolar subepithelial layer may be noted.

directing the point of the trocar slightly outward and upward. Without penetration into the foramen itself, a drop of 20 per cent solution of silver nitrate was deposited in the mucous membrane at this point, the cannula was reinserted and the trocar withdrawn. In this way, the remainder of the nasal mucosa was not injured. The rabbits were normal and were entirely free from snuffles.

Even though extreme care was exercised, numerous experiments were necessary because of the danger of subsequent respiratory infection. A nasal discharge was almost always present for from a few days to a week following the injection. In approximately half of the rabbits treated, even after an interval of two or three months, a purulent infection of the maxillary sinus was present. In a smaller percentage of cases there was some fatal pulmonary complication, demonstrable both macroscopically and microscopically.

The animals were killed at daily intervals for the first two weeks after the application of the silver nitrate and at weekly intervals thereafter, tissue from

the nose being obtained over a period of three months. This was removed from the point of injection, which was easily demonstrated in recently treated animals and fairly readily found even in animals that had been treated two or three months earlier, because in these the mucosa was grossly thicker and more fibrous and paler at the point of application of the solution than in the noses of normal rabbits. Paraffin sections were stained with hematoxylin and eosin.

Fig. 2.—Mucosa one week after application of 20 per cent solution of silver nitrate (× 200). Beginning regeneration of the epithelial layer may be noted. A single layer of epithelial cells overlies vascular granulation tissue in which cellular infiltration is marked.

COMMENT

Figures 1 to 5 reveal considerable cellular infiltration in the nasal mucosa following the application of silver nitrate. This was present to a considerable extent three months afterward. The mucous membrane was much thicker and more fibrous than normal. Certain degenerative

changes occurred even in the nerve fibers in response to the caustic action of silver nitrate (figs. 3 and 4). These changes may explain the relative freedom from hyperesthetic symptoms noted clinically.

Although the epithelium was largely regenerated (figs. 2 and 3), even to the point of the formation of cilia (fig. 5), after a period of three months it was pseudostratified, was still metaplastic in appearance

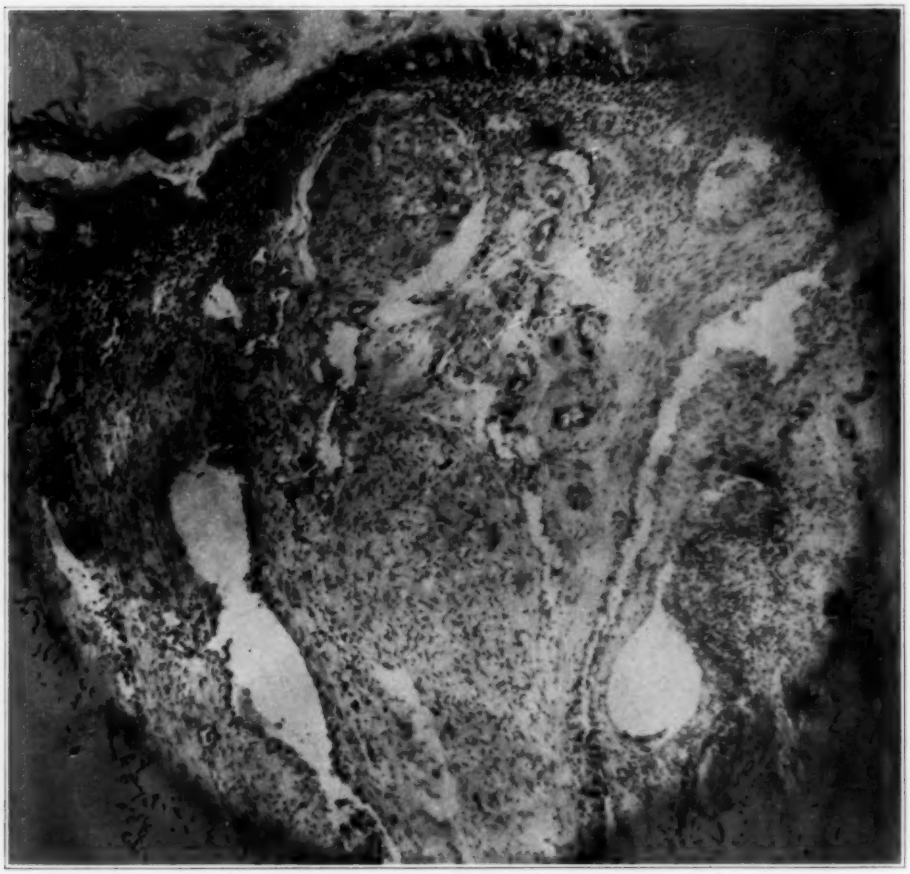

Fig. 3.—Mucous membrane four weeks after treatment with 20 per cent solution of silver nitrate (× 100). The epithelial regeneration is well marked. There are marked cellular infiltration and a great increase of connective tissue. Just beneath the layer of epithelial cells is a large nerve fiber in which there is a certain amount of cellular infiltration and vacuolization.

and contained more than the usual amount of goblet cells (fig. 5). The subepithelial glands had apparently regenerated, some of them as early as from four to six weeks after treatment (figs. 3 and 4). Beginning repair of the epithelium was noted as early as one week after treatment

Fig. 4.—Nasal mucous membrane six weeks after treatment with 20 per cent solution of silver nitrate (× 45). Marked fibrosis is present, involving even the blood vessels. The nerve fiber shows degenerative changes. The epithelium and glands have regenerated. Cellular infiltration is marked, particularly beneath the epithelium.

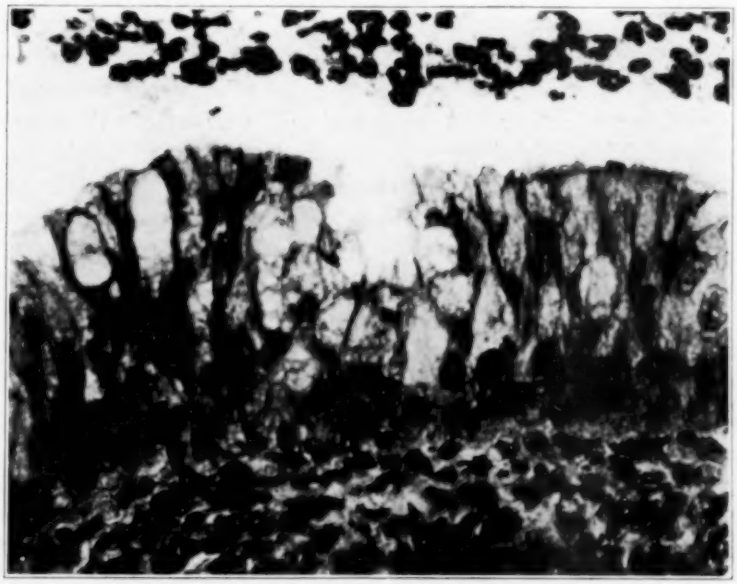

Fig. 5.—Nasal mucous membrane eleven weeks after treatment with 20 per cent solution of silver nitrate (× 425). This is the same section as that shown in figure 2. The regenerated epithelium, although containing cilia, is much more cellular than, and half again as thick as, normal. Goblet cells are abundant. The subepithelial tissue presents a marked contrast to the normal, in that cells are very abundant.

(fig. 2). In the subepithelial layers there were rather marked cellular infiltration and a considerable increase in connective tissue cells (figs. 3 and 4).

The subject of repair of the mucous membrane of the nose and paranasal sinuses has been considered in various articles in the last few years. It has been generally believed that healing of the lining of the nose and sinuses occurred by the formation of scar tissue. Recently, several observers have published results which tend to substantiate this belief;[1] others have shown that complete regeneration of the mucosa may occur.[2]

Following curettage of the lining of the antrum of dogs[2d] and of man,[2e] it was found that the mucous membrane was completely regenerated in from three to five months. The regenerative process in the nasal and antral epithelium proceeds rapidly; Beck noted evidence of it one week after the nasal mucosa had been cauterized with chromic acid, and McGregor saw evidence of regeneration sixteen days after operation on the antrum. Others, too, have noted regeneration of the epithelium of the antrum, but have found that the underlying tissue does not return to normal even after a year;[1a] instead it is either more cellular fibrous tissue, in which there is lack of regeneration of the glands and cavernous system,[1a] or firm scar tissue.[1b]

SUMMARY

Following the application of solution of silver nitrate to the nasal mucous membrane of rabbits, the epithelium begins to regenerate as early as one week after treatment, and cilia may appear three months afterward. This epithelium does not appear normal, but is pseudostratified and contains an abnormal number of goblet cells. The underlying tissue is fibrous and thick, and, although it contains glands which

1. (*a*) Matwejew, D. N.: Das Regenerationsvermögen der Nasenschleimhaut nach operativen Eingriffen, Monatschr. f. Ohrenh. **63**:1293 (Dec.) 1929. (*b*) Semenov, Herman, and Kistner, F. B.: Repair in the Paranasal Sinuses of Man Following Removal of the Mucous Membrane Lining, Proc. Soc. Exper. Biol. & Med. **27**:322 (Jan.) 1930.

2. (*a*) Beck, O.: Histologische Untersuchungen über die Wirkungen der Chromsaüre auf die Nasenschleimhaut, Ztschr. f. Hals-, Nasen- u. Ohrenh. **15**:395, 1926. (*b*) Dutheillet de Lamothe, G.: Les injections sclérosantes, traitement de choix des hypertrophies de la muqueuse nasale, sans malformations osseuses sous-jacentes, Rev. de laryng. **51**:657 (Nov. 30) 1930. (*c*) Gorham, C. B., and Bacher, J. A.: Regeneration of the Human Maxillary Antral Lining, Arch. Otolaryng. **11**:763 (June) 1930. (*d*) Knowlton, C. D., and McGregor, G. W.: How and When the Mucous Membrane of the Maxillary Sinus Regenerates: An Experimental Study in the Dog, Arch. Otolaryng. **8**:647 (Dec.) 1928. (*e*) McGregor, Gregor: Further Proof of the Regeneration of Mucous Membrane in the Human Antrum, Arch. Otolaryng. **14**:309 (Sept.) 1931.

have apparently regenerated, it is not normal in appearance, but is characterized by marked cellular infiltration (figs. 3 and 4). Perhaps of greater importance in connection with the study of vasomotor rhinitis is the fact that the nerve fibers show cellular infiltration and vacuolization. These changes in the nerve fibers may explain the beneficial results obtained in the treatment of vasomotor rhinitis by the topical application of silver nitrate solution to the membranes in the region of the nasal ganglion.

A COMPREHENSIVE ROENTGEN EXAMINATION OF THE PARANASAL SINUSES

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NEW YORK

The keynote of this presentation may be summarized in the terse question, "Is the referring physician getting sufficient and accurate information from the average roentgen examination of the paranasal sinuses?" I believe not. I frequently review and confer on films made elsewhere, and find the majority of them to be sadly deficient in many respects. The films are frequently of poor quality and taken with obviously poor or antiquated technic. Many should not have been accepted for interpretation. The reports frequently show a lack of comprehension of the subject, and conclusions are frequently wrong and without foundation. It is obvious that the progress in this special field of roentgenology is not taken advantage of in the average examination, and, in consequence, valuable information is lost and specialists are frequently ill advised. It is, therefore, my intention to outline the examination that I have used for the last two years.

The importance of precision of technic in the making of the roentgenograms cannot be stressed sufficiently. Concepts of "normals" develop in the minds of both the otolaryngologist and the roentgenologist, which are invaluable and indelible, but years of experience and knowledge of pathology are all to no avail if the technic is not perfect in every detail, to assure uniform films of the best quality. There must be no blurring due to motion. The placing of the patient's head and the angle of the central ray must never vary for the particular position. In other words, films are acceptable only without flaws; otherwise they must be rejected. The same high standards prevail for acceptable roentgenograms of the sinuses as for other types of specialized roentgenography. I would therefore formulate the following as a minimum standard for an acceptable roentgenographic examination of the paranasal sinuses:

1. Six is the minimal number of exposures, so as to permit the study of each sinus or group of cells in at least three different projections.

2. There must be no evidence of motion in any of the films.

3. At least one film, preferably the fronto-antral, should be taken in the erect position, and in this position the floor of the antrum should

Presented at the Clinical Conference of the Park East Hospital, New York, May 19, 1932.

not be overlapped (occasionally unavoidable) by the postcranial fossa or the petrous temporal bones.

4. Orbital projections of the ethmoid cells are indispensable, for they furnish the only views obtainable of these cells without objectionable superimposed shadows.

TECHNIC

A description is now in order of the important features of the technic of the examination. All exposures are made with the dental cone, long distance technic, which assures small diaphragming and easy focusing, with nearly parallel rays.

Fig. 1.—Postero-anterior, prone exposure of the ethmoid cells. The sphenoid sinus and most of the ethmoid cells are projected through each other, so that negative shadows of pneumatic normal cells obliterate positive shadows of pathologic cells and vice versa. See figure 4, which shows no superimposed pneumatic structures and is consequently a true representation of the condition of the ethmoid and sphenoid cells.

This secures a wealth of detail equal to that of the Potter-Bucky diaphragm, without the disadvantage of long exposures and the difficulty of angle work with the latter.

Exposure 1 (fig. 1) is a prone, postero-anterior projection of the sinuses and is labeled "ethmoids." The forehead and the nose touch the cassette, and the center ray is focused so as to make an angle of 10 degrees with the perpendicular toward the feet. This roentgenogram gives a concept of the general condition of the ethmoid cells, and affords a view for comparison with the others. Its value for

ethmoid cells alone is distinctly limited, but the exposure has supplementary value in the examination, and should not be excluded. It is indispensable when stereoscopic studies are made.

Exposure 2 (fig. 2) is a slightly modified "Granger" exposure. The patient is prone with his head placed as for exposure 1. The center ray is now directed toward the top of the head at about an angle of 7 degrees with the perpendicular. This exposure supplements exposure 1 to make a serviceable stereoscopic pair, if so desired. Although the technic that I advocate varies slightly from that recommended by Granger, the variation is negligible as regards the roentgenographic results, but it affords a much more comfortable position for the patient, which

Fig. 2.—Modified "Granger" exposure with clearly defined roof (Granger line), floor and walls. Pneumatic cells with clearly defined septums are present throughout. The findings are confirmed by an "orbital" exposure (fig. 4), in which both sides of the sphenoid sinus are clearly shown as pneumatic and normal.

fact is of the utmost importance in eliminating motion in the roentgenograms. All patients can be posed with ease and immobilized perfectly. The Granger exposure procures a postero-anterior projection of the sphenoid sinus through the lower portion of the frontal sinus, and shows the sphenoid cells more clearly than in any other postero-anterior view. The roof of the sphenoid sinus, called the "Granger line," when clearly defined with pneumatic cells beneath it, excludes exudate in the sphenoid cells. The sinus can usually be made out in its entirety, and pathologic changes, in the whole or a part, are instantly apparent to the experienced

observer. Some roentgenologists express preference for the occipitomental exposure. However, this is not procurable in stout or ill patients and does not compare favorably at all with the Granger exposure for uniform, clear detail. The Pfahler method of placing a special large film in the mouth is an improvement on the occipitomental exposure, but offers to a less extent the same objections. The sphenoid sinus must also be studied in the orbital and lateral views. This must be a routine procedure, for frequently anatomic variations and pathologic processes make it necessary to obtain all possible information from all three views before any opinion can be advanced.

Fig. 3.—True lateral, prone exposure. This is of value principally for anatomic observations of the depth of the sinuses and the thickness of their walls, to be considered in the interpretation of the other views.

Exposure 3 (fig. 3) is a true lateral exposure of the sinuses and is usually taken with the patient prone on the table. This film is of particular importance because it reveals the anatomy of the sinuses and indicates what to expect in the other views. The depth of the frontal, sphenoid sinuses and antrums and the thickness of the anterior and posterior plates of the frontal bone and of the floor of the anterior fossa of the skull are all factors of prime importance and must be determined before the interpretation of the films is attempted.

Exposures 4 and 5 (figs. 4 and 5) are orbital projections of the ethmoid cells and afford views of these structures with the minimum of overlapping shadows. The credit for this exposure is due to Reiss as its originator. However, the lack

of a definite technic which can be relied on to give constant results has been responsible for its tardy general adoption. After several weeks of experimentation in the laboratory, a simple technic was evolved which permits an effective way of securing these exposures satisfactorily and uniformly. Perfect orbital foramina and oblique projections of the sphenoid cells and frontal sinuses are also obtained, and are most useful in supplementing the other studies. These exposures show all of the cells within the ethmoid capsule, with an absolute minimum of superimposed bony shadows and practically no superimposed pneumatic structures. A mastoid block-out is used on an 8 by 10 inch (20.3 by 25.4 cm.) cassette. It is placed transversely on the table, under the patient's face. To make the orbital projection of the right ethmoid capsule, the center of the right eye is placed directly over the left (with respect to the patient) window of the block-out. The nose and forehead touch the cassette, and the long diameter of the head is held perpendicular to the cassette. The dental cone is used, as in all the other exposures, and directed to the

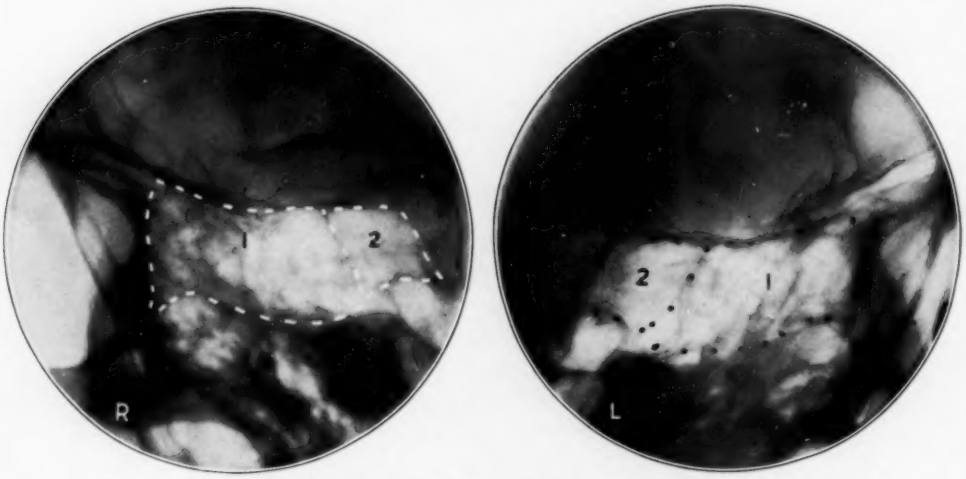

Fig. 4.—Orbital projections of ethmoid and sphenoid cells with the elimination of superimposed pneumatic structures. These views are most important for securing information about the ethmoid cells and sphenoid sinus. *1.* Cells within the ethmoid capsule. *2.* Sphenoid cells. The anterior cells on the right side are pneumatic but clouded, with slightly thickened septums. All the other cells throughout are normal.

center of the fenestra at an angle of 30 degrees to the right side and 25 degrees to the feet of the patient (20 degrees to the feet in children). The distance used is 24 inches (60.9 cm.). The technic is reversed as regards left and right to secure the orbital projection of the left ethmoid cells.

Exposure 6 (fig. 6) is for the frontal sinuses and antrums, and is always taken in the erect position. This determines the presence and amount of freely movable fluid in the antrums or frontal sinuses. It permits air to collect in the upper part of the sinuses to form a contrasting background for exposing the contents of the sinuses. The exact thickness of granulations and intrinsic tumors shows clearly in this exposure and may be estimated accurately. The petrous temporal bones and the posterior fossa of the skull should be projected below the floor of the antrums whenever possible, so as to avoid confusing superimposed shadows. In

this conjunction, I wish to submit a photograph of a simple, inexpensive film-holder for the erect position, which facilitates this exposure with a genuine economy of time and effort (fig. 7).

INTERPRETATION

Of primary importance in the interpretation of the films is the recognition and reporting of unusual anatomic variations. I am reminded of a discussion, some time ago, with a prominent otolaryngologist, who insisted that the left frontal sinus was pathologic because it was opaque. As a matter of fact, the sinus was unilateral in its development, and the specialist was convinced only after a demonstration of

Fig. 5.—Postero-anterior exposure in the erect position for the frontal sinuses and antrums. The frontal sinuses are clear, pneumatic, with clearly defined walls and normal. The left antrum is pneumatic, with clearly defined walls, and normal. The right antrum is approximately one-fourth filled with freely movable fluid. The prone positions showed irregular, slight clouding. The presence of fluid was not demonstrable in the prone exposures. The clearness of definition of the walls of the right antrum is brought out by the contrasting background of air above the fluid level, clearly indicating the absence of thickened membranes or granulations. In other words, an acute or subacute infection is present. Contrast this with the thickened antral walls in figs. 7, 8 and 9.

the lateral exposure, which showed the thick bone on the left side, projected through the pneumatic right side. Similarly, a few weeks before this paper was presented I reviewed some films on which a report had been submitted of unilateral disease of the frontal sinuses. Here the lateral view showed one side of the sinus to be shallow, the other

quite deep. The ignoring of this anatomic variation was responsible for the error. The thickness of the plates of the frontal bone must be weighed in the balance before interpreting films of the sinuses. Clearness of definition of the walls of the sinuses and cells is almost as important as radiolucency. The recognition of blurring and loss of detail of bony markings is important and usually indicates chronic infection with granulations or thickened membranes, possibly with polypoid degeneration, but the following exception should be borne in

Fig. 6.—A simple, inexpensive, cassette-holder for erect roentgenography, made from a discarded tube stand. It permits easy and rapid posing of the patient and centering of the roentgen rays. The use of the small diaphragm, long distance technic obviates the use of the more expensive, cumbersome grid apparatus, securing as good if not better detail.

mind: If the plates for the frontal bone, as viewed in the lateral projection, are fused above in a very acute angle, one cannot expect as sharp a delineation of the roof of the frontal sinuses as when the angle is obtruse or dome-shaped. Unilateral development of the frontal sinuses will influence the appearance of the sphenoid sinus in the Granger exposure and cause a relative opacity on the undeveloped side.

Here, the checking up in the orbital and lateral views, as a rule, settles this question decisively. The depth of the antrums, as shown in the lateral projection, is also important and permits anticipating the degree of radiolucency to be expected in the postero-anterior exposures. The significance of these anatomic considerations cannot be stressed sufficiently.

With a proper concept of normal roentgenograms, making due allowance for anatomic variations, it is possible to state almost as a routine, the exact pathologic condition present and differentiate between

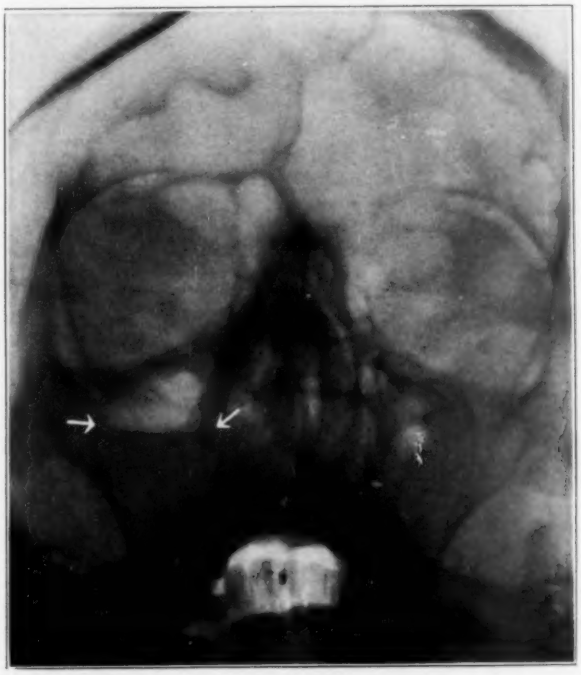

Fig. 7.—The frontal sinuses are large, clear, pneumatic, with clearly defined cells and septum, and normal. The right antrum is almost completely opaque, and contains dense mucopus. The shadow did not vary with a change in position. The left antrum is slightly more than half filled with movable fluid and clearly shows the fluid level. Above the fluid level, in the pneumatic portion of the antrum, the air forms a contrasting background, permitting clear visualization of the roof and walls of the antrum. They are distinctly thickened from the presence of dense granulations, and measure from one eighth to one quarter of an inch in thickness.

slightly thickened membranes, granulations and their thickness (fig. 9), tumors (fig. 7) and movable fluid (figs. 7 and 9). With improvement in technic, the indications for the use of radiopaque mediums are constantly diminishing. The exposure in the erect position assures gravitation of fluid to the floor of the antrums and frontal sinuses, permitting

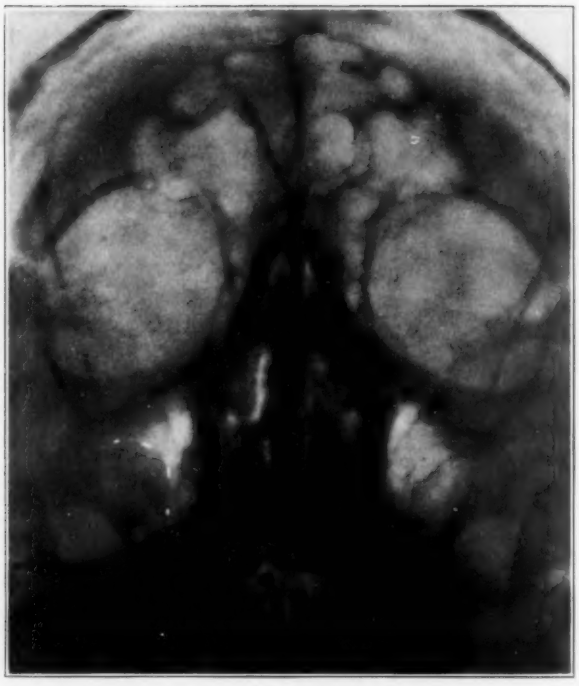

Fig. 8.—The frontal sinuses are large, symmetrical, pneumatic, with clearly defined walls, and normal. The right antrum is slightly clouded, with slightly thickened walls, and contains granulations, but no free fluid. The left antrum is also clouded and contains a large, globular polypoid mass on its external wall. The walls are markedly thickened and measure almost half an inch (1.2 cm.) in thickness.

Fig. 9.—The left frontal sinus contains freely movable fluid. The walls and roof are thickened, with clouding above the fluid level. The circumscribed opacity and fluid levels are seen in the erect position only. The left antrum shows dense granulations over three eighths of an inch (0.9 cm.) thick, but no movable fluid.

the air above the fluid level to act as a contrasting background, showing the pathologic changes in clear relief. If the erect exposure is not included in the examination, the old noncommittal formula of "granulations or pus" is the best interpretation that can be suggested. If the entire sinus is opaque, my experience would indicate the presence at most times of dense mucopus, and the examination should be repeated after irrigation and aspiration of the sinus; then, in most instances, detailed information is obtained as to the condition of the antral walls and the presence or absence of tumors. Should this procedure fail to yield more information, the use of radiopaque mediums is definitely indicated, for it is impossible to differentiate further the opaque shadows without this help. When radiopaque mediums are used, injections should not be

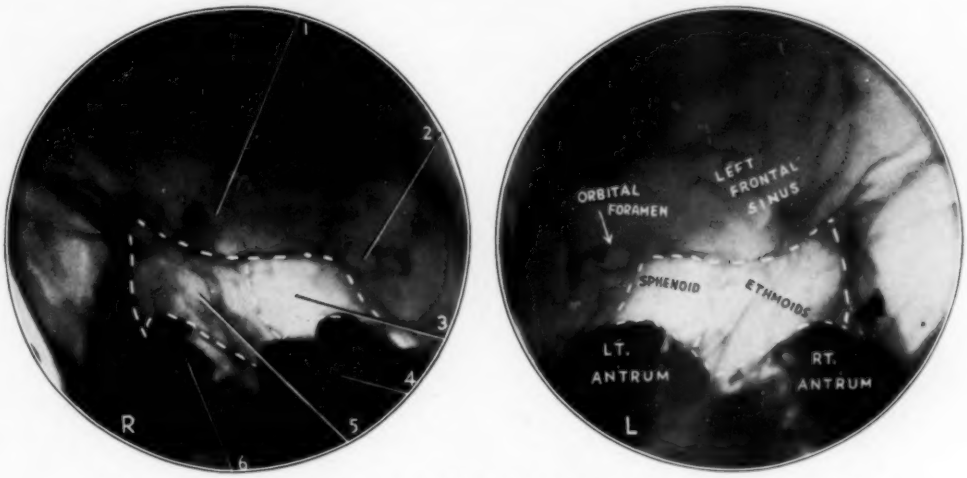

Fig. 10.—Orbital views of the right and left ethmoid cells. *1.* Right frontal sinus. *2.* Right orbital foramen. *3.* Right side of the sphenoid sinus. *4.* Right antrum. *5.* Right ethmoid cells. *6.* Left antrum. Both antrums have received an injection of iodized poppy seed oil 40 per cent, indicating the relation of the antrums to the ethmoid and sphenoid cells, in the orbital projection. The left ethmoid cells are pneumatic, with clearly defined septums, and normal, whereas the extreme anterior cells on the right side are clouded or opaque and contain granulations or exudate. The upper posterior cells on the right side are pneumatic and normal, whereas the lower posterior cells are clouded or opaque, with markedly thickened walls, and contain granulations, possibly exudate. Both the right and left sides of the sphenoid sinus are normal. The orbital foramina are clearly seen. As this exposure can be reproduced repeatedly without any variation, it may be used to determine the size of the optic foramen. The frontal sinuses are also visualized, projected obliquely.

made into both sides at one time, otherwise conflicting superimposed opaque shadows result, which confuse rather than clarify the picture. At least postero-anterior and lateral views in both the prone and the

erect positions should be made, utilizing gravity to reach all parts of the sinuses or cells. In this way no constant filling defects can be overlooked, and at most times valuable additional information is obtained.

Some experience is essential in acquiring confidence in the interpretation of the orbital projections of the ethmoid cells. In an acceptable roentgenogram, the details of the cells should be nearly as good as those in a mastoid process (figs. 5, 10 and 11). Normal ethmoid cells are not the rule in the vicinity of Greater New York, but are encountered sufficiently often to maintain my absolute confidence in this exposure. Opaque or clouded or pneumatic cells, rarefied or thickened septums can be uniformly differentiated and located (figs. 10, 11 and 12).

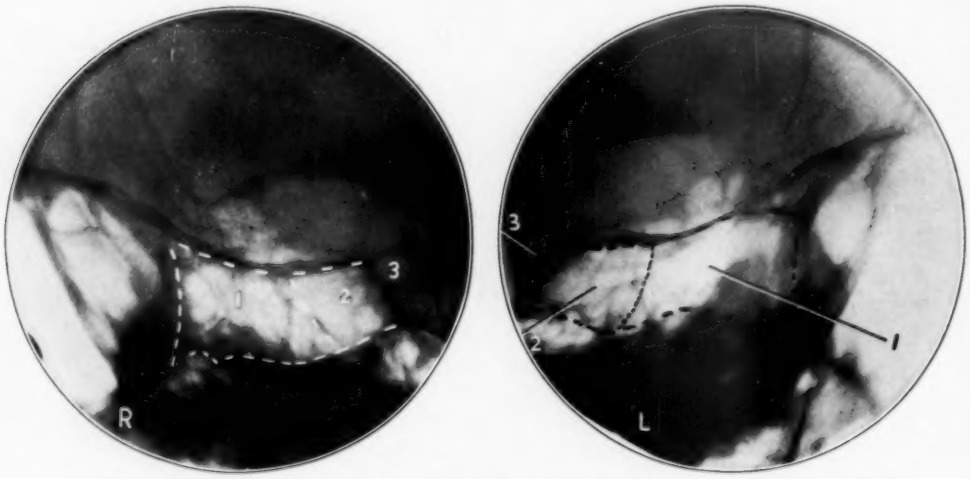

Fig. 11.—*1.* Right ethmoid cells. *2.* Right sphenoid cells. *3.* Right orbital foramen. The orbital view of the right ethmoid cells shows pneumatic cells throughout with a clear definition of the septums. There are no evidences of disease, whereas the orbital projection of the left ethmoid cells shows the upper anterior cells to be slightly clouded from thickened membranes, the lower anterior cells to be opaque from exudate and the posterior cells to be pneumatic, with clearly defined septums, and normal. The sphenoid cells are pneumatic, with clearly defined walls, and normal on both sides. Frequently, considerable doubt may be entertained as to the condition of the ethmoid and sphenoid cells from the postero-anterior and lateral studies, which cannot be cleared up without the help of the orbital views (figs. 12 and 13).

Experience with this exposure teaches the unreliability of the postero-anterior and lateral views, with their confusing overlapping shadows.

Certain supplementary observations should be made as a routine by the roentgenologist, as they may have some bearing on the condition of the sinuses or the symptoms. Blurring and loss of detail within the nasal chambers in a person with a history of long-continued symptoms

is usually indicative of the presence of polypoid tissue and suggests the presence of a similar condition in the paranasal cells and sinuses. Syphilis has been detected twice by the presence of discrete gummas in the frontal bone, and Paget's disease three times by its characteristic blotchy bony texture. Evidences of increased intracranial pressure is frequently noted and suggests further examination for intracranial tumors. The condition of the sella turcica and clinoid processes, as observed in the lateral and Granger exposures, should be noted as a routine, for they occasionally present abnormalities of marked signifi-

Fig. 12.—The postero-anterior ethmoidal and lateral exposures were totally confusing, with irregular clouding throughout the cells on both sides, making it impossible to determine the condition of the groups of ethmoid and sphenoid cells. This exposure shows dense opacity of the right side of the sphenoid sinus, with obliteration of the Granger line (roof) of the sphenoid sinus, indicating the presence of dense granulations or exudate, probably both, whereas the cells on the left side of the sphenoid sinus are pneumatic, but very slightly clouded. This is confirmed by figure 13, in which the individual involved ethmoid and sphenoid cells are clearly indicated. Compare with figure 2.

cance. Intra-oral dental films should be made to determine possible dental origin in every instance of antral disease, when the other sinuses are normal. A careful history should be taken in each case and due

consideration given facts which may have some bearing on the findings, and it should not be forgotten that it is of prime importance to confer with the surgeon in any case of doubtful character. A comprehensive report should include (1) a brief description of the anatomy, particularly when variations are present, and their bearing on the interpretation, (2) a description of the findings, indicating pathologic conditions or abnormalities, (3) conclusions and (4) advice for supplementary examinations if the conclusions are doubtful.

Stereoscopic films in routine work are unnecessary. Stereoscopic lateral views supplementing exposures 1 and 2, taken as a stereoscopic pair, give valuable studies in three dimensions when so desired. There are, however, specific indications for them, in the study of tumors or

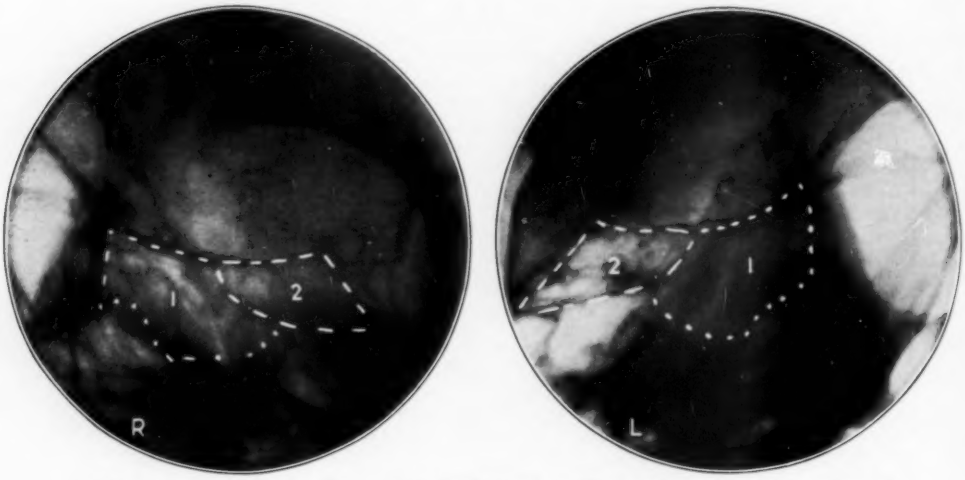

Fig. 13.—*1.* Ethmoid cells. *2.* Sphenoid cells. The orbital view shows general clouding of the ethmoid cells on the right side with markedly thickened septums. Granulations are present with exudate in some of the lower central and posterior cells. The orbital view of the left ethmoid cells shows general opacity throughout. The right side of the sphenoid sinus is observed to be opaque, whereas the left side is pneumatic but very slightly clouded from thickened membranes. The findings confirm those in the Granger exposure (fig. 12), and prove the futility of attempting to diagnose the condition of ethmoid and sphenoid cells without these indispensable exposures.

severe trauma. I have found stereoscopic studies of particular value in the location of some groups of pathologic cells with respect to the ends of probes, to guide the surgeon in further operative procedures in obstinate cases. For the convenience of the referring physician, films should be labeled properly and completely, for each film shows certain of the sinuses better than the others. The films should also be blocked out, so as to eliminate the glare of light coming through the unimportant parts of film and prevent unnecessary eye strain.

SUMMARY

1. A minimum of six exposures is essential for a comprehensive roentgen study of the paranasal sinuses, permitting each sinus or group of cells to be studied in at least three projections.

2. Variations in technic are outlined to standardize and popularize these exposures.

3. The fronto-antral exposure must be made in the erect position with the elimination, whenever possible, of superimposed shadows of the petrous temporal and posterior fossa of the skull.

4. Films showing traces of motion or faulty posing must be rejected.

5. Anatomic variations must be given proper consideration in the interpretation of the roentgenograms.

9 West Sixty-Eighth Street.

PRIMARY SUBMUCOUS LARYNGEAL ABSCESSES

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Acute purulent and nonpurulent conditions of the larynx were formerly designated in many different ways. The following terms were used indiscriminately: edema laryngis, edema glottidis, laryngitis submucosa, laryngitis edematosa, laryngitis phlegmonosa, laryngeal abscess, angina laryngea infiltrata, laryngitis seropurulenta and angina laryngea edematosa. Kuttner [1] emphasized that the process currently termed laryngeal abscess, as well as a majority of the processes designated by the foregoing terms, arise from similar conditions, and that therefore the majority of these terms are misleading and superfluous. Most contemporary writers agree on this, and the generally accepted opinion now is that abscess formation in the larynx follows a breaking down and pus formation in acute submucous laryngitis. Any irritation such as an acute catarrh, trauma due to a foreign body, or talking too much will first cause an acute submucous laryngitis. Infections following influenza, typhoid fever, scarlet fever, tuberculosis and syphilis can also cause such a submucous inflammation of specific origin, and all may be followed by abscess formation. In many cases the perichondrium is involved immediately after the onset of the submucous laryngitis, and through this involvement is separated from its cartilage. The inflammatory process then pushes against the adjoining parts and appears as a swelling of these tissues, so that, as in many of the cases reported in the literature, the abscess mass breaks through the laryngeal cartilage and appears as a so-called external abscess. The abscess may also point into the lumen of the larynx, causing stenosis, or else, if the portions of the thyroid cartilage adjoining the piriform fossa are involved, may point into the pharynx. The latter course occurs in but few cases, and will be further described among the operative cases presented here.

Small abscesses superficially located in the larynx are the type most frequently found. They first appear as an edematous swelling and later fluctuate. They are generally situated on the epiglottis or on the arytenoid cartilages. Innumerable cases of this type are described in the

1. Kuttner, A.: Das Larynxoedem und die submucoese Laryngitis, Virchows Arch. f. path. Anat. **139**:117, 1895.

literature: Chiari,[2] Schrötter,[3] Imhofer,[4] von Heymann,[5] Levinstein,[6] Kuttner,[1] Jackson,[7] Hecht,[8] Tobold,[9] Verweyen [10] and Cruveilhier.[11]

Discrete abscesses in the vocal cords are rare, but have been described (Chiari [2a]). They usually appear as circumscribed, yellowish, soft swellings, which can easily be opened and cause little trouble.

The so-called prelaryngeal abscess is a special form of laryngeal abscess and develops after infection of the prelaryngeal lymph node. Occasionally such abscesses form following intralaryngeal operations, especially operations on the ventricular bands. Occasionally one sees them after such operations on the tuberculous larynx. Hoensch [12] described three such cases. The infection probably spreads along the intralaryngeal lymphatics. Békiarian [13] described eighteen cases of prelaryngeal abscess and claimed that the majority are "cold abscesses," and are associated with an advanced tuberculosis of the larynx. We have observed such an infection of the prelaryngeal node with abscess formation on several occasions, and also following intralaryngeal manipulations. They do not, though, appear to be very common. Such a case was observed in the Sea View Hospital, New York, following electro-cauterization of the interarytenoid space in a tuberculous larynx. The so-called external laryngeal abscesses seem to be more common, developing on top of the aforementioned acute submucous laryngitis, usually as a palpable, fluctuating mass on the surface of the thyroid cartilage, when the inflammation erodes through this cartilage. Less common, but also more serious, are the circumscribed submucous abscesses

2. Chiari, O.: (*a*) Chirurgie des Kehlkopfes und der Luftroehre, Stuttgart, Ferdinand Enke, 1916; (*b*) Ueber primaere acute Entzuendung des submucoesen Gewebes des Kehlkopfes, Wien. klin. Wchnschr. **10**:109, 1897.

3. Schrötter, L.: Vorlesungen ueber die Krankheiten des Kehlkopfes, Vienna, W. Braumüller, 1892, p. 99.

4. Imhofer, R.: (*a*) Larynxabszesse, Ztschr. f. Ohrenh. **76**:36, 1917; (*b*) Metastatische Larynxabscesse im Verlauf einer Pyaemie, Internat. Zentralbl. f. Laryng. u. Rhin. **31**:116, 1915.

5. von Heymann, Paul: Handbuch der Laryngologie und Rhinologie, Vienna, A. Hölder, 1898.

6. Levinstein, Oswald: Pathologie und Therapie der Epiglottitis abscedens. Ztschr. f. Laryng. **8**:310, 1916-1919.

7. Jackson, Chevalier, and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929, p. 844.

8. Hecht: Zur Lehre von den Kehlkopfabszessen, Thesis, Würzburg.

9. Tobold: Larynxabszess, Berl. klin. Wchnschr. **1**:39, 1864.

10. Verweyen, Theodor: Ueber Larynxabszess, Würzburg, Becker, 1881.

11. Cruveilhier: Anatomie pathologique du corps humain, Paris, J.-B. Baillière, 1829, vol. 1, book 2, fig. 1.

12. Hoensch: Prälaryngeale Abszessbildung nach endolaryngealen Operationen, Ztschr. f. Ohrenh. **71**:286, 1914.

13. Békiarian, Ajot: L'abscès froid prelarynge, Thesis, Paris, 1907-1908.

pointing into the lumen of the larynx, which frequently cause dangerous stenoses. Such abscesses can form in the mucous membrane of both the thyroid and cricoid cartilages. They are often difficult to treat. At times they show nothing more than a bulging of the subglottic region parallel to the vocal cords. There are also isolated abscesses of the piriform fossa, which really belong to the group of superficial laryngeal abscesses previously mentioned. Such a case was described by Miller.[14] The last type to be considered consists of those abscesses which appear clinically as isolated abscesses of the piriform fossa, but which are in reality of greater extent; that is, they form in the mucosa of the thyroid cartilage and spread into the fossa piriformis as being a locus minoris resistentiae.

Such abscesses also give rise to difficulties in treatment. The majority of laryngeal abscesses occur in adults. That they never occur in children, as was formerly believed, can no longer be maintained. Descortes[15] described twenty-nine cases in children. Ballin[16] maintained that the abscesses occur chiefly on the left side, but our observations as well as the cases described in the literature do not support this.

DIFFERENTIAL DIAGNOSIS

A differential diagnosis can be made accurately only by direct or indirect examination of the larynx. Edema of the larynx is more or less semitranslucent. The abscess may have the appearance of a tumor or of a syphilitic or tuberculous lesion. By palpation one may find fluctuation when the abscess is found on the outside of the larynx or, when the abscess is extrinsic, for instance, in the piriform fossa. A cyst may at times resemble an abscess. Aspiration of the suspected lesion may confirm the diagnosis of the abscess.

SYMPTOMS AND COMPLICATIONS

The symptoms are at first scratching in the throat, then pain on speaking and swallowing, especially if the abscess is located on the epiglottis. In the lumen of the larynx the abscess can easily cause stenosis and hoarseness. If the abscess forms during an acute infectious disease, there are usually fever and generalized weakness, but cases have been described in which the local and general symptoms were so slight as hardly to have been noticed by the patients. In some cases the abscess ruptures spontaneously (Verweyen[10]), especially if it is super-

14. Miller, J. W.: Paralaryngeal Abscesses; Two Cases, Bull. Beth Israel Hosp., 1930, no. 4, p. 260.

15. Descortes, René: Les abscès laryngés chez l'enfant, Thesis, Paris, 1912-1913.

16. Ballin, M. J.: Laryngeal Abscess, New York M. J. **104**:781 (Oct. 21) 1916.

ficial. Abscesses on the laryngeal surface of the epiglottis and in the lumen of the larynx can lead to sudden fatal stenoses. Cases also have been described (Chiari [2]) in which the infection spreads to the lymphatics of the neck and mediastinum, or in which through aspiration of the pus from the abscess, pneumonia developed. A generalized sepsis also occurs at times. That the reverse is true, namely, a laryngeal abscess forming hematogenously from a sepsis, is demonstrated by Imhofer's case,[4b] perhaps the only one of its kind reported in the literature.

TREATMENT

The superficial abscesses on the epiglottis and the arytenoids and the very rare isolated abscesses of the vocal cord can easily be opened with a scalpel and heal without complications. It sometimes happens, though, that the perichondrium and the cartilage are involved and pieces of the latter and occasionally the whole arytenoid slough out, with irreparable damage to the larynx and permanent hoarseness as a result. The external abscesses in the presence of fluctuation over the thyroid or cricoid cartilages usually heal after incision from without, provided that there is not too great a necrosis of the cartilage. In such a case, especially when the cricoid is involved, the laryngeal structure may collapse, and through scar tissue lead to a severe stenosis requiring long, tedious treatment by dilation.

Carrari [17] described a laryngeal abscess following typhoid, in which tracheotomy and four other operations were needed to overcome the stenosis which followed the healing of the perichondritis. In intralaryngeal abscesses with obstruction of the larynx and in the presence of dyspnea, tracheotomy should not be postponed, and if the abscess does not fully empty after an intralaryngeal incision, laryngotomy should also be performed to prevent a widespread destruction of the laryngeal cartilages. Most writers stress that only a prompt tracheotomy can protect against unpleasant surprises. Sudden death due to laryngeal stenosis was noted chiefly in the prelaryngoscopic era. In all probability George Washington's death in 1799 was due to such an unrecognized laryngeal abscess. Although laryngoscopic examination was unknown in his time, several authors who made a study of this statesman's illness came to the conclusion that his death resulted from asphyxia brought on by an abscess of the larynx: Green,[18] Jackson [19] and Freudenthal.[20]

17. Carrari, G. G.: Duplice complicanza laryngea da tifo abdominale, Gior. med. osp. cov. Venezia **3**:120, 1929.

18. Green, Horace: A Treatise on Diseases of the Air Passages, New York, G. P. Putnam, 1846, p. 32.

19. Jackson, James: Memoirs on the Last Sickness of General Washington and Its Treatment by the Attending Physicians, Boston, privately printed, 1860.

20. Freudenthal, W.: Unusual Instances of Laryngeal Abscesses, Laryngoscope **21**:1083, 1911.

The cases which we are about to describe require special attention in regard to therapy, as they are unusual in both their clinical course and the treatment indicated. In our perusal of the literature we found no similar case described. We could not, therefore, make use of any generally accepted measures of treatment or of any specially described in the literature as applicable to such cases and we were forced to devise a special method which we feel justified in describing here, since we now consider it to be the method of choice in such laryngeal abscesses.

REPORT OF CASES

CASE 1.—Miss A. R., aged 14, in October, 1930, complained of hoarseness of several months' duration. She stated that she had had a cold a few months previously, and had had increasing hoarseness since then. There was no pain on

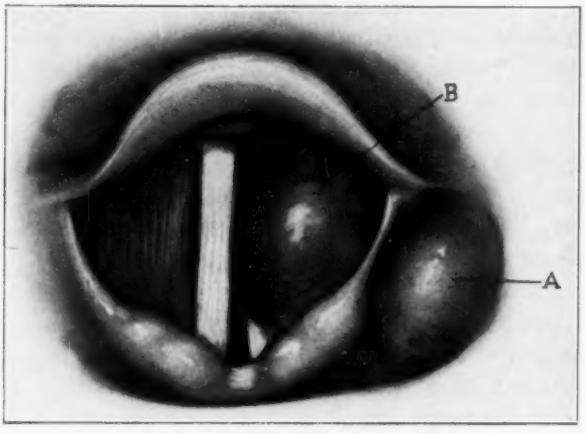

Fig. 1.—Abscess of the larynx pointing into the piriform sinus (*A*) and into the ventricular band (*B*).

swallowing, and no dyspnea. In general, she felt well. Laryngoscopic examination revealed edematous swelling of the entire left piriform fossa and a similar thickening of the left ventricular band, so that the vocal cord was almost entirely hidden (fig. 1). However, the function of the vocal cords was apparently normal. General examination gave negative results. Roentgenograms of the lungs and lateral views of the larynx revealed nothing. The Wassermann test was negative. With the aid of a mirror and a guarded laryngeal knife, an incision was made into the mass in the piriform fossa, releasing about 5 cc. of thick yellow pus and with some immediate relief of the hoarseness. Bacteriologic examination showed Streptococcus hemolyticus. The patient was seen at weekly intervals, and it was noted that the abscess in the piriform fossa refilled repeatedly. As a result, the abscess was reopened about eight times, sometimes indirectly with a bent laryngeal knife, and on several occasions through the Jackson laryngoscope with a straight knife, and at each instance the same amount of pus was released. During the second month following the last incision, the patient's voice was clear and the swelling was slight. In May, 1931, however, the patient returned, and the entire

piriform fossa was involved with accompanying swelling of the left ventricular band and marked hoarseness. Our operative procedure leading to a cure will be described later.

CASE 2.—Mr. C. B. O., on Oct. 21, 1925, complained of pain in the throat and difficulty in swallowing for several weeks. Examination of the larynx showed swelling of aryepiglottic fold and left piriform sinus. A diagnosis of laryngeal abscess was made, and incision through the direct laryngoscope released a large amount of foul pus. Bacteriologic examination showed Staphylococcus aureus. The swelling, however, did not entirely disappear and incision had to be repeated, in all about eight times, on each occasion the same amount of pus being released. The patient was seen regularly and the swelling seemed much smaller, but occasionally it would increase in size. At times a small amount of pus could be seen

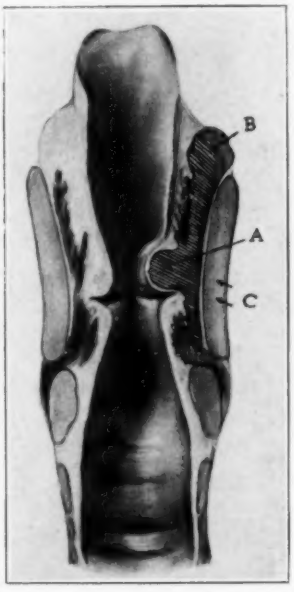

Fig. 2.—A section through the larynx to show the supposed extension of the abscess: *A*, the abscess pushing into the lumen of the larynx; *B*, the abscess bulging into the piriform fossa, and *C*, arrow showing the site of the window made through the cartilage in order to approach the abscess.

oozing out of a small fistula. In June, 1926, the patient returned with the same symptoms as in the beginning, and examination revealed the same area to be much swollen, involving the left arytenoid cartilage.

It is evident that the two cases resemble each other in the laryngoscopic picture as well as in their clinical course. We were sure of the diagnosis of laryngeal abscess and also realized that superficial incisions would not result in a cure. We therefore had to assume that the abscess was situated much more deeply, apparently in the inner part of the larynx. We also assumed that the abscess had formed in the submucosa of the thyroid cartilage in the interior part of the larynx and

had pointed upward, into the piriform fossa (fig. 2). In view of the otherwise good condition of the larynx and the absence of dyspnea, we hesitated to use the procedure advocated in the textbooks for laryngeal abscess, that is, making a laryngeal fissure. We therefore decided to attempt an approach and drainage of the abscess from the outside by creating a window in the thyroid cartilage. The same procedure was used in both cases. The thyroid cartilage was exposed as figure 3 shows, and an opening the size of a one cent piece made with a sharp curet. The perichondrium of the interior part of the larynx bulged

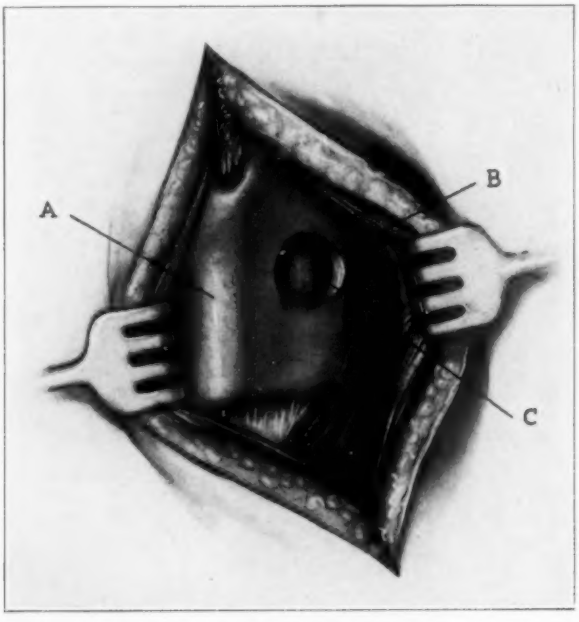

Fig. 3.—*A*, thyroid cartilage; *B*, window in the thyroid cartilage to approach the abscess through the inner perichondrium (*C*).

out into the opening and was fluctuant. An incision into the perichondrium was made and about 10 to 15 cc. of thick creamy pus released in both cases. This proved that the abscess was situated between the inner perichondrium of the thyroid cartilage and the mucosa of the larynx. A rubber drain was inserted. The wound was kept open for several weeks by means of a drain and was completely healed within the next three weeks.

Both patients were seen one year after the operation, and the larynx then was found to be entirely normal with a scar on the outside over the thyroid cartilage.

CONCLUSIONS

A general survey of primary laryngeal abscesses is given with their pathology, symptomatology and treatment.

The rarest kind of laryngeal abscess is an abscess in the mucosa of the thyroid cartilage extending into the piriform fossa. Such cases do not respond to simple incisions into the bulging mass in the piriform fossa.

A method is described for approaching such abscesses externally via a window resection of the thyroid cartilage.

ERYSIPELAS OF THE PHARYNX AND LARYNX

BENJAMIN KATZ, M.D.
LOS ANGELES

A state of confusion exists in the literature related to this clinical entity, since different authors, taking into consideration single components of the symptom complex in question, describe typical erysipelas of the larynx under various names.[1]

From a clinical point of view, erysipelas of the pharynx and larynx forms two different groups. In the first one, described by Darluc,[2] Ryland,[3] Radcliffe[4] and Cornil,[5] the disease spreads from the skin to the mucous membranes. In the second group, the pharynx and larynx are primarily affected by the infection (Massei[6]).

ETIOLOGY

For a certain time Fehleisen's[7] streptococcus was considered to be the specific germ of erysipelas. But Fraenkel[8] proved that this organism was identical with the common pus-forming streptococcus. Fraenkel obtained a pure culture of streptococcus from a case of purulent peritonitis at autopsy and inoculated a rabbit with it. A typical picture of erysipelas developed in the animal's ear, which was confirmed by histologic examination. However, the question of specificity of this infection is not settled as yet, as two schools with different opinions exist.

Read before the Los Angeles County Medical Association, April 21, 1932.

1. Hubbard, T.: An Unusual Type of Laryngeal Edema, Tr. Am. Laryng., Rhin. & Otol. Soc. **20**:287, 1914. Richards, L. G.: Acute Laryngeal Obstruction, J. A. M. A. **95**:766 (Sept. 13) 1930. McIntosh, R., and Nichol, K. D.: Abscess of the Larynx in Infants, ibid. **90**:2095 (June 30) 1928.

2. Darluc: J. de méd. et de pharm., July, 1757.

3. Ryland, quoted by Kuttner, Arthur: Larynxödem und submucöse Laryngitis, Berlin, G. Reimer, 1895.

4. Radcliffe: Severe Erysipelas of Head and Face Producing Edema of the Larynx, Lancet **1**:536, 1835.

5. Cornil, V.: Arch. gén. de méd. **19**:257 and 444, 1862.

6. Massei, F.: Ueber das primäre Erysipel des Kehlkopfs, Berlin, A. Hirschwald, 1886.

7. Fehleisen: Die Aetiologie des Erysipels, Berlin, T. Fischer, 1883.

8. Fraenkel, E.: Zur Lehre von der Identität des Streptococcus pyogenes und Streptococcus erysipelatus, Centralbl. f. Bakt. **25**:1265, 1889.

Birkhaug,[9] by means of agglutination and other sero-immunologic methods, Tunnicliff,[10] by cultures on chocolate agar, and Dick and Dick,[11] by the method of soluble toxins, found that the streptococci of erysipelas are immunologically specific and distinct.

In contradiction to the views of this school stands the aforementioned experiment of Fraenkel, corroborated by other observations. Kilkbride and Wheeler, Zlatogorov and Derkatch, McLachlan, McCann, Park and Spiegel [12] have found that cross-neutralization between erysipelas toxin and scarlet fever antitoxin or between scarlet fever toxin and erysipelas antitoxin could be obtained. In an epidemic of so-called septic sore throat, which, according to Davis,[13] is caused by a specific Streptococcus epidemicus, Williams [14] neutralized the toxins from the same streptococcus (Davis) by serum produced from a scarlet fever strain of streptococcus, while erysipelas, as Davis stated, was a frequent complication of the epidemic.

The foregoing observations demonstrate that the sero-immunologic reactions of different types of streptococci are interchangeable.

It is well known that in the clinical course of erysipelas the same micro-organism can produce phlegmon or an abscess as well as an extension of the infection, thus showing that different clinical entities can be produced by the same microbe. A similar conclusion can be drawn from the observations of Dick and Dick,[15] who obtained a culture of hemolytic streptococcus from the cutaneous lesions of erysipelas, and, by inoculation of the throats of volunteers, produced acute angina, resembling ordinary sore throat without the manifestations of erysipelas on the skin. On the other hand, acute tonsillitis is often a starting point of erysipelas on the mucous membranes, with subsequent extension to the skin.

This evidence demonstrates that the clinical manifestations produced by a certain type of streptococcus vary in accordance with the reaction of the system, not being governed merely by specific characteristics of the particular germ.

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9. Birkhaug, K. E.: Erysipelas: V. Observations on the Etiology and Treatment with Erysipelas Antistreptococcic Serum, J. A. M. A. **86**:1411 (May 8) 1926.

10. Tunnicliff, R.: Streptococci from Scarlet Fever, Erysipelas and Septic Sore Throat, J. A. M. A. **94**:1213 (April 19) 1930.

11. Dick, G. F., and Dick, G. H.: Specificity of Soluble Toxins Produced by Hemolytic Streptococci, J. A. M. A. **93**:1784 (Dec. 7) 1929.

12. For reference to these authors, see Dick and Dick [11] and Fraenkel.[8]

13. Davis, D. J.: Septic Sore Throat, J. A. M. A. **93**:978 (Sept. 28) 1929.

14. Williams, A., in discussion of Davis.[13]

15. Dick, G. F., and Dick, G. H.: Experimental Sore Throat, J. A. M. A. **89**: 1135 (Oct. 1) 1927.

Relapses in erysipelas comprise another subject of controversy, the discussion revolving around the specific and nonspecific origin of the phenomenon in question. Durham [16] explained them by a sudden liberation of toxic substances during the active phase of secondary invaders caused by a great variety of micro-organisms, to which no antibodies were produced during the primary attack of erysipelas; this explanation leaves out the single specific germ as a cause of relapses. Birkhaug [17] suggested that a state of bacterial allergy to Streptococcus erysipelatis is a possible explanation of relapses within previously sensitized areas.

Before all of the aforementioned and many other controversial points pertaining to the one hundred and two different human pathogenic streptococci (Shwartzman [18]), eighteen strains of which belong to the group of Streptococcus erysipelatis, are cleared up by further investigation, it is worth while for the clinician to remember the statement of Gottstein,[19] that the form of the disease caused by streptococci depends on the part of the body affected and the reaction of the organism.

THE NATURE OF SEPTIC SORE THROAT

The epidemics of so-called septic sore throat (Davis), considering the source of infection, the way of transmission, the clinical complications and the bacteriologic resemblance of the infection, are identical with those which have been definitely reported as milk-borne epidemics of erysipelas of the throat. In the epidemic of sore throat and erysipelas in England described by Hunter, Hall and Lawson,[20] erysipelas was transferred through milk from a farm the owner of which was sick with erysipelas. Similarly, in the epidemic of septic sore throat mentioned by Davis, the infection originated in the throat of a milker and was disseminated through the milk to the consumers, in many of whom sore throat and erysipelas of the skin developed.

There is a striking correspondence between both infections from a bacteriologic point of view. The intensive studies of Williams [21] demonstrate that strains from erysipelas show growth characteristics similar to those described as belonging to the strains of epidemics of septic sore throat.

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16. Durham, H. E.: A Theory of Relapses, J. Path. & Bact. **7**:240, 1901.

17. Birkhaug, K. E.: Erysipelas: VIII. Bacterial Allergy to Streptococcus Erysipelatis in Recurrent Erysipelas, J. A. M. A. **90**:1997 (June 23) 1928.

18. Shwartzman, G.: Studies on Streptococcus Bacteriophage, J. Exper. Med. **46**:497 (Sept.) 1927.

19. Gottstein, A.: Erysipelkokken, in Leibreich, Oscar: Encyklopaedie der Therapie, Berlin, A. Hirschwald, 1897, p. 225.

20. Hunter; Hall, and Lawson: An Epidemic of Sore Throat and Erysipelas, Glasgow M. J., October, 1890.

21. Williams, A., in discussion of Pilot, I., and Davis, D.: Sporadic Septic Sore Throat, J. A. M. A. **97**:1691 (Dec. 5) 1931.

SOURCES OF INFECTION AND MODUS OF INVASION

Studying the question of from what source the infection reaches the mucous membrane, Gerber [22] found that the infection may come from the dust of the atmosphere or from direct contact, as through food, swabbing of the throat and other means.

As contributing factors to the invasion of the infection may be mentioned all conditions which produce congestion of the mucosa, such as colds and tonsillitis. However, in certain instances erysipelas starts without preliminary inflammation, the portal of entry being the physiologic spaces (*Intercellularlucken*) between the cells of the epithelium of the mucosa, from which the infection spreads to the lymphatic apparatus of the root of the tongue and then may affect the larynx.

If erysipelas of the pharynx and larynx develops as a secondary process, the infection spreads from the skin to the mucosa in the following ways: (1) through the lips into the mouth; (2) through the nasal passages; (3) through the external meatus, the middle ear and the eustachian tube, and (4) through the conjunctiva and the saccus and ductus lacrimalis.

PATHOLOGIC ANATOMY

Pathologically, erysipelas of the pharynx and larynx is nothing more than an acute edematous swelling of the mucosa (Virchow [23]). A proof of this opinion can be found in the fact that on the skin, in places where it is thin and delicate and the connective tissue is loose, as on the eyelids and praeputium, erysipelas manifests itself more by edematous swelling than by the usual infiltration. Besides edema, pathologically, erysipelas is characterized by a large accumulation of streptococci in the lymphatic vessels, sometimes to a complete obliteration of them (Fraenkel).

If proliferation of the formed elements develops, with a consequent transformation of the serous exudate into a purulent process, the process clinically undergoes a change into phlegmon or an abscess. "Erysipelas of the pharynx and larynx, as of other parts, often is the first stage of a process, which later may be transformed into the condition of phlegmon" (Gerber).

SYMPTOMS

The disease starts with a fever of from 102 to 104 F., and is characterized by a rapid onset and progress, sometimes after previous tonsillitis, or primarily affecting the larynx. Since the infection spreads through the lymphatics of the root of the tongue, the tongue becomes

22. Gerber: Acute infectiose Phlegmone, in von Heymann, Paul: Handbuch der Laryngologie und Rhinologie, Vienna, A. Hölder, 1896, p. 1265.

23. Virchow, R.: Verhandl. d. Berl. med. Gesellsch. **1**:80, 1887.

swollen, causing dysphagia. When the process spreads to the larynx, the mucosa and the submucous connective tissue rapidly become filled with serous exudate, swell and form a stenosis. The rapid formation of an inflammatory edematous swelling with rapid extension to the surrounding parts ("wandering") is typical of erysipelas of the larynx.

There are three types of erysipelas of the mucosa: (1) that with inflammatory redness, (2) that with the formation of vesicles and (3) that with subsequent necrosis. The first form is characterized by a bright red discoloration of the mucosa with a shining glassy appearance. In the second form (erysipelas bullosa), the size of the vesicles varies from that of a pea to that of a cherry. In the third group, the mucosa may undergo necrosis, with the production of a characteristic odor.

DIAGNOSIS

All edemas of the larynx of a noninflammatory character which occur in the course of systemic and constitutional diseases can be easily excluded by the fact that erysipelas is an acute infection with a high temperature and general malaise, and by the absence of constitutional signs of the ailments mentioned. Acute edematous laryngitis of an inflammatory nature sometimes occurs as a reaction to the deep caustic action of chemicals, to contusions, to fractures of the bony skeleton of the larynx and to the presence of foreign bodies. In such cases a definite etiology of trauma is apparent, and, particularly, the picture of an acute infectious disease with a rapid onset, chills and temperature is absent. Acute laryngitis does not have such a rapid course, high temperature or pain on swallowing; the edema of the larynx characteristic of erysipelas is not present, and there is no stenosis. Acute secondary laryngitis during the course of such infectious diseases as measles, scarlet fever and typhoid fever can be excluded by the absence of a primary causative illness. In phlegmon of the larynx the swelling is not as rapid as in serous, movable edema, but is of a slowly progressive type due to infiltration by formed elements, with subsequent purulent liquefaction of the infiltrate.

The following case, which I reported elsewhere,[24] exemplifies primary erysipelas of the larynx. In this case a combination of treatments with a polyvalent streptococcus serum and vaccine therapy was employed, with good results.

In M. Sch., a physician, aged 31, acute edema of the larynx with stenosis and dyspnea developed following acute tonsillitis.

The previous history was of no special interest. The internal organs were normal. There was a bright redness of the entire pharynx. The mucosa of the larynx was inflamed and of a bright red color with a glassy shine. There was

24. Katz, B.: Erysipelas of the Larynx, Yezhemïes. Ushn. Gorhov. i Nosov. Boliezn. **8**:379, 1913.

edema of the epiglottis, both arytenoids and the false vocal cords. The rima glottidis, owing to edema, was half the regular size. The true vocal cords could not be seen; the voice was hoarse and dull. There was dyspnea, with stridor and dysphagia. The glands of the neck were swollen and painful.

During the evening and at night, after admission, swabbing of the larynx with epinephrine solution brought temporary relief. Attempts to insert O'Dwyer's tubes failed, because they were expelled by coughing. Ice internally and cold applications to the neck did not bring relief. The edema of the larynx gradually increased. At noon of the following day (December 4), a tracheotomy was performed. The next day a typical erysipelas developed around the wound; the edema of the larynx progressed. On December 6, two doses of 100 cc. of polyvalent antistreptococcus serum were injected. The next day the doses were repeated and on December 8, 100 cc. of the same serum was injected. On December 9, the temperature became normal. The edema of the larynx subsided to such an extent that the patient could breathe without the tracheotomy tube, which was removed. However, four days later a peritonsillar abscess developed, and about 1 teaspoonful of pus was evacuated on puncturing the abscess; edema of the larynx and fever returned, and the tube was reinstalled. During the next three weeks, the patient had a few more relapses and became weaker. To combat the relapses, 2,500,000 of streptococcus vaccine was injected on January 9. After one day the temperature became normal. On January 14, the edema of the larynx and the erysipelas on the skin had completely subsided. On January 16, the tracheotomy tube was removed, and a week later the patient was discharged.

PROGNOSIS

The prognosis depends on three factors: (1) Danger from erysipelas per se. Symmers and Lewis [25] reported a mortality equal to 10.1 per cent. (2) Danger from stenosis. In 168 cases of stenosis, 140 deaths occurred, 83 per cent. (3) Complications of the disease. If erysipelas spreads to the lungs, the mortality is 100 per cent.

TREATMENT

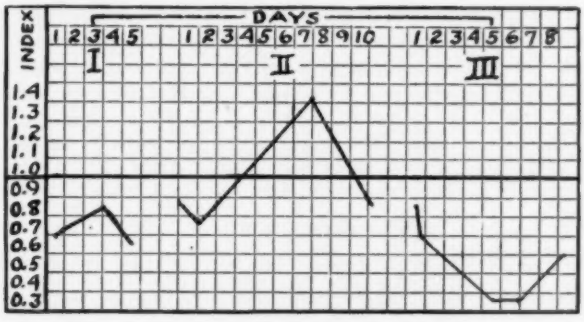
In the beginning of the disease, cracked ice in the mouth, cold applications to the neck and local applications of solution of epinephrine are indicated. If edema of the larynx develops, prompt tracheotomy is imperative. A proper diet and care of the heart should be carried out in the usual manner.

Besides these symptomatic measures, a specific therapy, namely, serotherapy and vaccinotherapy, should also be applied. The patient in the case reported received 100 cc. of a polyvalent type of serum intramuscularly five times and 2,500,000 of streptococcus vaccine. The rationale of this treatment is that the serum neutralizes the toxins while the vaccine stimulates the formation of antibodies. Birkhaug recommends a specific erysipelas antistreptococcus serum which he injects intramuscularly, the dose being 100 cc. of unconcentrated and 15 cc. of

25. Symmers, D., and Lewis, K. M.: The Antitoxin Treatment of Erysipelas, J. A. M. A. **89**:880 (Sept. 10) 1927.

concentrated serum. The average total dose in children is 26 cc. of concentrated serum (McCann [26]).

The theoretical foundation for the use of vaccine in erysipelas was laid by Ruediger, who proved that the opsonic index in this disease is greatly diminished. The injection of vaccine increases the opsonic index, but only under certain conditions. Mathews [27] showed that if the injected dose of vaccine is too small (chart), the immediately obtained increase in the opsonic index is rather insignificant in regard to the power and duration. If a medium dose is injected, the index, immediately after injection, becomes slightly lower (negative phase), but immediately afterward continually increases for a period of seven days and then gradually decreases. If the dose is too large, following the



Action of different doses of vaccines on the opsonic index in erysipelas. Curve I is for small doses; curve II, for medium doses, and curve III, for larger doses.

injection a pronounced drop of the index occurs, which continues for a few days and only slowly rises toward the starting level. Therefore, the more beneficial dose is the medium one.

SUMMARY

Erysipelas of the pharynx and larynx presents a definite clinical and pathologic entity, a diagnosis of which can be made without manifestations on the skin. If acute edema of the pharynx or larynx develops, with fever and general malaise, subsequent to or during acute tonsillitis or a cold, the disease should be considered as erysipelas, if streptococci are the causative germ. The milk-borne epidemics of sore throat were definitely reported by some as epidemics of erysipelas of the pharynx

26. McCann, W. S.: The Serum Treatment of Erysipelas, J. A. M. A. **91**:78 (July 14) 1928.

27. Mathews: Lancet **2**:926, 1908; quoted by Wolfson: Ueber Vakzinationstherapie, in Wolff-Eisner, A.: Handbuch der Serumtherapie und experimentellen Therapie, Munich, J. F. Lehmann, 1910.

and larynx. The so-called septic sore throat, considered from the standpoint of the source of infection, modus of transmission, complications and bacteriologic resemblance, is identical with erysipelas of the throat. Treatment, in addition to symptomatic measures, should include serotherapy and vaccinotherapy.

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MUCOSA OF THE MAXILLARY SINUSES OF THE RABBIT

PRODUCTION OF EXPERIMENTAL EOSINOPHILIA BY STIMULATION OF THE NERVE AND BY TRAUMA

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Vagotonia has been associated with allergy by some clinicians in spite of the lack of definite proof. Eppinger and Hess [1] first formulated the conception of vagotonia and sympatheticotonia. They observed that if pilocarpine or atropine produced a strong parasympathetic reaction, the injection of epinephrine was without effect on the sympathetic nerves. Likewise, if epinephrine produced a strong sympathetic reaction, pilocarpine did not stimulate or atropine paralyze the parasympathetic nerves. On the basis of these findings, they concluded that clinical cases showing a functional imbalance between the two sets of nerves could be classified as vagotonia or sympatheticotonia, depending on which system was hyperactive. They stated that autonomic imbalance need not involve the entire autonomic system, and also stated that vagotónia occurred locally. It was emphasized that, in their opinion, an exaggerated tonus in one division of the automonic nervous system precluded heightened reactivity in the other division.

Eppinger and Hess [1] also regard eosinophilia as a sign of vagotonia. Eosinophilia may be associated with nasal allergy, bronchial asthma, urticaria and intestinal parasites. Evidence that eosinophilia in persons with intestinal parasites is associated with overstimulation of the parasympathetic system is shown by the occurrence of increased peristalsis and spastic intestinal contractions. The spasm in bronchial asthma can be relieved by the administration of atropine, which diminishes parasympathetic tonus, or by the administration of epinephrine, which increases sympathetic tonus.

Stimulation of the vagus nerve in laboratory animals does not reproduce allergy as recognized in man, but it does tend to exert an influence on the eosinophil count in the blood stream. Bertelli and his co-workers [2]

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1. Eppinger, H., and Hess, L.: Zur Pathologie des vegetative Nervenssystems, Ztschr. f. klin. Med. **66**:345, 1909; **68**:205, 1909.

2. Bertelli, Falta and Schweeger: Ueber die Wickselwirkung der Drüsen mit innerer Sekretion, Ztschr. f. klin. Med. **71**:23, 1910.

observed that stimulation of the parasympathetic system with drugs produced mononucleosis and eosinophilia in the blood stream. Stimulating the sympathetic system with epinephrine caused a drop or even disappearance of eosinophils with an increase in neutrophils. Schwenker and Schlecht [3] confirmed the findings of Bertelli, except that they found no rise in percentage of eosinophil cells in the blood stream with drugs stimulating the parasympathetic system.

Hajos and his co-workers [4] found that in a series of ten guinea-pigs with exposed vagus nerves, stimulation with the faradic current for from one-half hour to eight hours caused a rise in the blood eosinophilia of six guinea-pigs. The operative procedure itself had no effect. Smith and Benner [5] reported a case in which the administration of digitalis caused a rise in blood eosinophilia varying from 9 to 30 per cent. This was thought to be due to a vagus effect. Atropine in doses of 1 mg. caused a drop, and in seven hours the blood eosinophilia was still below the usual level. Epinephrine in doses of 0.3 cc. caused a sharp drop with a return to the usual level. These authors quote the nasal findings of Dr. L. W. Dean, who was called in for a rhinologic consultation. He found no evidence of allergy in the nose on examination, but a marked eosinophilia was seen in smears taken from the nose.

In view of the work done apparently demonstrating the relationship of vagus stimulation and blood eosinophilia, it was thought that it would be of interest to determine if vagus stimulation would cause a tissue eosinophilia. A series of rabbits about 6 months of age was given pilocarpine hydrochloride intravenously in doses of 4 mg. per kilogram of body weight. The maxillary sinus mucosa was removed following injection at varying intervals of from several minutes to twenty-four hours. It was found that a maximum eosinophilia occurred in approximately five minutes, and this disappeared in twenty-four hours. In a series of five rabbits killed five minutes after the intravenous injection of pilocarpine, one showed a marked, one a moderate, and two a slight eosinophilic infiltration and one none. Hematoxylin-eosin and Giemsa stains were employed. The Giemsa stain is helpful in distinguishing pseudo-eosinophils from true eosinophil cells of the rabbit. The Giemsa stain brings out the red granules of the true eosinophil cells, but leaves the pseudo-eosinophil cells stained as the usual neutrophil cell.

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3. Schwenker, G., and Schlecht, H.: Ueber den Einfluss sympathetica und autonomatroper Substanzen auf die Eosinophillen Zellen, Ztschr. f. klin. Med. **76:** 77, 1912.

4. Hajos, K.; Nemeth, R., and Enyedy, Z.: Ueber den Einfluss directen Vagusreizung auf die Eosinophilie und auf die Leberstructure, Ztschr. f. d. ges. exper. Med. **48:**590, 1926.

5. Smith, A. E., and Benner, S. R.: Eosinophilia Due to the Administration of Digitalis, Am. Heart J. **7:**182, 1931.

The explanation of this phenomenon of tissue eosinophilia is rather difficult, but it may be due to a disturbance in ionic equilibrium in the tissue attracting eosinophil cells by chemotaxis from the blood stream. Some work to support this assumption may be found in a paper by Howell and Duke.[6] They found that stimulation of the vagus caused an increased output of potassium in the perfused heart. They attributed the vagus stoppage to potassium which by stimulation of the vagus nerve is assumed to be converted from a nondiffusible to a diffusible form.

Hetenyi and Goal [7] found that there was an increase of blood calcium on the administration of pilocarpine, but since the blood serum becomes thickened after its administration the results were not conclusive.

The question arose as to the importance of the time factor in the experiments reported here. According to the findings of Newkirk,[8] who investigated the action of pilocarpine on excised small intestine of the rabbit immersed in warm oxygenated saline solution, the addition of pilocarpine caused a great increase in tonus which slowly decreased, but if the pilocarpine solution was changed for a fresh saline solution, a second rise in tonus occurred as the alkaloid diffused out of the cells into the saline. If the saline was immediately changed, however, for the original pilocarpine solution so that diffusion ceased on account of an equal concentration inside and outside the cells, the tonus also disappeared. It seems that the characteristic effect appeared only during the time that the pilocarpine was in the act of passing through the cell membrane.

The irrigation of the maxillary sinus with alizarin according to the technic described by Wenner [9] causes an infiltration of eosinophil cells into the sinus mucosa. This effect is augmented under certain conditions as reported by Wenner and Nemours.[10] It was thought that pilocarpine would also augment the effect of alizarin. A series of five

6. Howell and Duke: Experiments on the Isolated Mammalian Heart to Show the Relation of Inorganic Salts to the Action of the Accelerator and Inhibitory Filters, J. Physiol. **135**:131, 1907.

7. Hetenyi, G., and Goal, A. W.: Die Blutkalkregulation im menschlichen Organismus. Wirkung von Adrenalin, Atropin und Pilocarpin, Ztschr. f. d. ges. exper. Med. **75**:516, 1931.

8. Newkirk, P.: Physiologische Wertbestimmung und Dünndarm, Arch. f. d. ges. Physiol. **147**:153, 1912.

9. Wenner, W. F.: The Effect of Calcium Precipitating Substances on the Ciliated Epithelium of the Maxillary Sinuses, J. Lab. & Clin. Med. **16**:341, 1931.

10. Wenner, W. F., and Nemours, P. R.: The Mucosa of the Maxillary Sinus of the Rabbit: The Effect of Acidosis and Alkalosis in the Changes in Ciliated Epithelium and Eosinophilic Infiltration Produced by Calcium Precipitating Substances, Arch. Otolaryng. **15**:270 (Feb.) 1932.

rabbits was given nasal irrigations with 0.5 per cent sodium alizarin sulphonate solution every forty-eight hours over a period of ten days. Immediately after each nasal irrigation, pilocarpine was given intravenously, 4 mg. per kilogram of body weight. The results of this experiment showed three rabbits with the usual response of eosinophils to irrigation with alizarin, and one with a moderately increased response to this dye; one rabbit presented the most intense eosinophilia I have observed in experimental eosinophilia of the maxillary sinuses. The lack of uniformity of the results may be expected, for this is perhaps due to the fact that in the majority of rabbits the effect of pilocarpine had disappeared at the time of autopsy (twenty-four hours after the last irrigation).

It occurred to me to note the effect of capillary constriction on the production of tissue eosinophilia by means of pilocarpine. Into a series of five rabbits was injected the usual dose of pilocarpine, and one minute after solution of pituitary in doses of 1 cc. was then given intravenously. The rabbits were killed five minutes later. The pituitary solution caused an almost total lack of eosinophils in the sinus mucosa.

The maxillary sinus mucosa removed from normal rabbits shows few or no eosinophils. It was thought that it would be of interest to study the effect of trauma and exposure in relation to eosinophilic response, if any. A series of five rabbits was prepared according to the following technic. Under sodium amytal anesthesia, a longitudinal incision was made on top of the head extending nearly to the tip of the nose. The skin was retracted and the periosteum cut on a lateral side and lifted over. A mastoid gouge was then used to uncover the bone above the maxillary sinus on one side; the bone covering the other maxillary sinus was left intact in order to have one side as a control. The exposed membrane could be seen to move up and down with the respiratory currents, and it was also noted that the respiratory rate was disturbed during manipulation of the sinus mucous membrane. The regular slow breathing stopped, and in some instances artificial respiration was required for a short interval. Apparently a strong inhibitory nerve reflex took place. The mucosa was loosened from the side walls as much as possible without causing perforation; then the excess blood was sponged away, and the periosteum pulled over the sinus. The final step was skin approximation. In twenty-four hours the animal was killed, and the exposed and control sinus membranes were sectioned. The control sinus showed no eosinophilia, but a marked eosinophilic infiltration was found in the exposed sinus mucosa. All five rabbits showed the same result. The question then arose: How soon did these eosinophil cells infiltrate the mucosa? It was found that in a series of rabbits prepared in the same way as in the first trauma experiment, and killed at varying lengths of time following exposure of the maxillary sinus

mucosa, a slight infiltration occurred about one-half hour after operation. In ten hours the result was a moderately large infiltration of eosinophil cells.

The most likely explanation of these results is probably in the stimulation of the endings of the vagus nerve in the mucous membrane. This would cause a change in ionic equilibrium and cause an eosinophilia by chemotaxis. Another possible explanation is the formation of eosinophils in a blood clot, as was pointed out by Liebreich.[11] He claimed to produce a large number of eosinophil cells in a clot in vitro by a special process of centrifugation and fixation. I know of no one in this country who has been able to duplicate his experiments. My sections had blood clots attached to them, and numerous eosinophils were also found in the blood clots as well as in the tissue. Just how they arrived there is open to question, but Liebreich's work is very suggestive.

CONCLUSIONS

1. Stimulation of the parasympathetic system with pilocarpine causes an infiltration of eosinophil cells into the maxillary sinus mucosa of the rabbit.

2. Trauma also causes the appearance of eosinophil cells into the mucous membrane, either due to stimulation of the vagus endings, or due to special changes taking place in the clotting of blood within the body, as pointed out by Liebreich.

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11. Liebreich, E.: Recherches morpho-biologiques sur le sang, Schweiz. med. Wchnschr. **2**:275, 1921.

SPONTANEOUS PERFORATION OF THE SIGMOID SINUS FOLLOWING THE MASTOID OPERATION

REPORT OF THREE CASES

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It has been my experience that some form of septic invasion of the lateral sinus occurs in slightly over 2 per cent of all suppurative mastoid inflammations (estimated in part). In one series of 646 operations on the mastoid, covering a period of ten years at St. John's Riverside Hospital, septic infections of the lateral sinus were observed 12 times. The incidence has been higher than this in cases of mastoiditis following contagious diseases, especially scarlet fever; also the prevalence has been greater in one year than in another, owing no doubt to the presence of more virulent types of micro-organisms in certain instances.

The question may be asked why such cases of spontaneous perforation of the sigmoid sinus should be segregated, as this complication results from a localized septic phlebitis involving the wall of the vessel, which eventuates in contamination of the blood stream by pyogenic bacteria and usually requires the treatment given in all such infections.

The reason is that certain of these cases afford an opportunity for noting the progress of the inflammation from day to day, thus permitting the observer to make deductions regarding the advance of infecting organisms through the wall of the vessel which would otherwise be impossible.

The three cases herein reported were so nearly identical in symptoms and pathologic process that for the sake of brevity they will be dealt with so far as possible as a group rather than individually. All occurred in children ranging in age from 5 to 7 years. As to the early history of the middle ear infections, one followed measles, one scarlet fever and one tonsillitis. Two cases involved a single mastoid and one both; two were cases of streptococcic infections and the causative organism in one is at this time unknown.

The operations on the mastoid uniformly revealed extensive bony necrosis necessitating laying bare the sigmoids, and in one instance a portion of the dura posterior to the sinus. After operation, the wounds did poorly and continued to suppurate, which was attributed to unusually

virulent types of organisms. In the bilateral infection in particular, which followed scarlet fever, the tissues died *en masse*. Within a few days the bone surrounding the wounds for a considerable distance appeared dead, and on one side there was a triangular area of dura covering the cerebellum which underwent a peculiar form of necrosis. It presently became dry, of a pale color and unusually transparent, somewhat resembling parchment in physical characteristics; through this window-like area pus could be seen covering the underlying structures. In this case the diagnosis of meningitis could be made from ocular inspection alone. (The patient ultimately died of general suppurative meningitis.)

The behavior of the sinus infections was similar in every instance. Also it should be stated that in the patient with scarlet fever both sigmoid sinuses became involved. The mode of onset was as follows: A few days after the operation on the mastoid a small, dark-colored, beadlike protuberance was seen jutting out from the sinus wall. This beadlike object was of course the inner coat of the vessel protruding through an ulcerated area in its outer wall. This protuberance was observed to increase in size for from two to four days, when rupture occurred followed by spontaneous hemorrhage from the sinus, which required firm tamponing to control. The necessity of keeping such wounds firmly packed is readily apparent. Politzer [1] said that "death from hemorrhage has been known to occur." The time between the operation on the mastoid and rupture of the sigmoid sinuses was on an average little more than a week. One side in the bilateral case ruptured at about eight days; the other, a few days later. In the unilateral cases rupture occurred on the sixth and the eighth day, respectively.

Following rupture in each instance the sigmoid sinus was slit open well beyond the confines of the necrosed area and packed with iodoform gauze, and the jugular vein was either resected or ligated. Under such treatment the patients in the unilateral cases recovered, but in each instance the convalescence was both stormy and protracted. In one case a metastasis developed in one foot; in the other pains in the joints and retinal hemorrhages occurred.

To me these cases presented some features of unusual interest. The first was the febrile reactions immediately following the operations on the mastoid, and in this connection only the two unilateral cases will be considered, as the bilateral case was complicated by meningitis. In each of these two cases there was a steady decline in temperature after the operation without fluctuations characteristic of contamination of the blood stream up to the time of rupture of the sigmoid sinus, despite

1. Politzer, A.: A Text-Book of Diseases of the Ear for Students and Practitioners, ed. 6, London, Baillière, Tindall & Cox, 1926, p. 598.

the fact that for approximately three days of this period an area in the vessel wall was so greatly thinned that only the inner coat protected the blood stream from the suppurating wound. It is true that at this time the temperature was still slightly elevated, but at no time was there any rise suggestive of septicemia. Almost immediately, however, following bleeding from the sigmoid sinuses (within a few hours), the temperature rose to nearly 104 F. in one case and to over 106 F. in the other, indicating that infectious material had suddenly been let loose in the circulation.

The absence of a fluctuating temperature prior to the time of rupture would seem to show with some degree of certainty that the intima offered in each instance a rather effective barrier against the passage of organisms into the blood stream; also it is worthy of note that so delicate a membrane further thinned by the stretching process could resist destruction for so long a period. Apparently in each of the four sinuses the lining of the vessel withstood the ravages of the wound infection as well as did the peripheral coat. It is likewise interesting that in none of the four sinuses affected was any thrombus in evidence, which also tends to support the conclusion that the infection had not penetrated to involve the endothelium prior to the time of rupture.

MacCallum,[2] in discussing the process of thrombosis of certain large vessels in the body, among which the cranial sinuses are enumerated, seems positive in the belief that traumatization of the endothelial lining makes possible the adherence of platelets to the vessel wall and permits the subsequent laying down of the various elements that go to make up the thrombus. The location of the injury naturally determines the site of the thrombus or at least its point of beginning.

In accordance with MacCallum's teaching, it is manifest that nature must always be at a disadvantage with an infection advancing from the periphery inward through the wall of an exposed sinus. Here the endothelium is the last stratum to be involved in the suppurative process, and until this structure is traumatized by the bacterial action, nature so to speak is not made aware of the approaching danger, nor is she placed in a position to offer defense by the erection of a thrombotic barrier. By the time bacterial action has caused sufficient roughening of the endothelium to permit of thrombus formation, and probably before this has occurred, the vessel wall is so weakened that rupture from pressure of the blood stream is inevitable.

Viewed in this light, it seems improbable that bacterial penetration from without through the wall of an exposed sigmoid can ever result in thrombus formation. It is my belief that whenever an area in the wall

2. MacCallum, W. G.: A Text-Book of Pathology, ed. 2, Philadelphia, W. B. Saunders Company, 1920, p. 13.

of an exposed sigmoid is eroded through by the action of an adjacent suppurative process, perforation of the vessel wall will invariably occur before a thrombus can form. As was previously intimated, such instances of spontaneous perforation of the sigmoid sinuses are in themselves of small concern to the otologist and would hardly be worthy of special mention except for the fact that they permit of ocular inspection and consequently of more accurate deductions regarding the passage of pathogenic organisms through the vessel wall than would otherwise be possible.

Unquestionably the mode of introduction of the infecting organisms into the lumen of the sigmoid is of importance, and it remains a matter of judgment to decide whether the findings relative to these four sinuses that lay bare in the wounds after operations on the mastoid warrant conjecture as to what probably happens in cases in which the vessel has not been exposed by operation.

To enumerate again the points of interest incident to the bacterial penetration through the walls of these four exposed sinuses, it may be stated that:

(*a*) The time required after operation varied from six to approximately eleven days.

(*b*) In two uncomplicated cases there was no evidence of contamination of the blood stream until a few hours after the sinus walls actually ruptured.

(*c*) In none of the four sinuses was any demonstrable thrombus in evidence.

(*d*) For approximately three days prior to rupture, each vessel wall showed a bulging area secondary to erosion of its outer coat.

(*e*) No appreciable thickening in the vessel walls capable of slowing the blood current was observed.

It seems probable to me that bacterial penetration through the wall of a sigmoid sinus covered by bony tissues differs little from the condition described. The time required might be somewhat less, but there is no apparent reason for believing that the walls of the sinus would be so thickened as to occasion a slowing of the blood current. If slowing of the blood current does occur, it is no doubt the result of compression from an accumulation of inflammatory products (perisinuous abscess). Nor can one assume that either thrombosis or blood stream infection takes place until the organisms have attacked the endothelial lining. As in the instance of the exposed sinus, one must believe that pyogenic organisms traversing the thickness of the sinus wall will of necessity cause an area of tissue degeneration.

Spontaneous hemorrhage as the result of peripheral invasion of an unexposed sinus is rare; this in itself is indicative of the fact that thrombosis does occur here. Why thrombosis occurs in the instance of the nonexposed and not in the exposed sinus is a matter of interest. The suggestion is offered that this can best be explained by the suppo-

sition that in the former instance the involved area in the vessel wall is supported by the overlying structures and that premature rupture of the intima is thereby prevented. Thus supported, the vessel wall may retain its integrity until the endothelial lining is sufficiently roughened by bacterial action to excite thrombus formation.

Having observed the rather great resistance offered by the wall of the sigmoid sinus against bacterial penetration, one may with some reason assume that blood stream infections by this route are probably less common than is generally supposed. In this connection it may be suggested that the discovery at operation of a ruptured sigmoid does not in every instance warrant the conclusion that the vessel wall has perforated in consequence of external erosion. On the contrary, careful observation rather frequently discloses evidence that bacterial activity within the vessel has been responsible for this occurrence. Since this article was written, the writer has seen two cases in which this was thought to have occurred.

In one, the sigmoid when uncovered at operation presented a glistening exterior; in fact, so far as could be observed, it was normal in appearance, but was on the point of rupturing. Perforation of this vessel wall occurred as the result of palpation in an effort to determine whether a thrombus was present, and a quantity of pus escaped into the wound.

The second case was almost similar to the first, differing only in the advancement of the suppurative process. At the time of the operation on the mastoid, the sinus plate was found intact and was thought to be healthy. The persistence of evidence of sepsis, however, demanded an exploration of the sigmoid sinus, and removal of the sinus plate a few days later disclosed the presence of a perisinuous abscess overlying a ruptured, suppurating sigmoid. There seemed to be hardly a doubt that the two cases were pathologically identical, the perisinuous abscess in the second instance having resulted from a rupture of the sigmoid in consequence of delayed operation.

In each instance the infecting organisms were believed to have gained entrance to the lumen of the vessel by thrombotic extension through a local tributary vein.[3]

CONCLUSIONS

1. Mastoid infections of exceptionally high virulency may cause complete degeneration of the tissue of an area in the sinus wall or in the dura, thereby producing pyemia in the one instance or meningitis in the other.

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3. Kopetzky, Samuel J.: Otologic Surgery, ed. 2, New York, Paul B. Hoeber, Inc., 1925, p. 283.

2. As regards suppurative inflammation attacking the wall of the lateral sinus from the peripheral side:

(*a*) The intima resists bacterial invasion to a remarkable degree.

(*b*) Without involvement of the endothelial lining of a vessel neither thrombosis nor infection of the blood stream can occur.

(*c*) In the instance of an exposed sinus, rupture of the vessel wall will almost inevitably occur before a thrombus can form.

(*d*) Infections by this route are relatively uncommon, comprising but a small part of the total number of infections of the sigmoid sinus.

(*e*) If the sigmoid sinus is supported by external structures, rupture may be delayed until a thrombus can form.

POSTDIPHTHERITIC LARYNGEAL STENOSIS

A REVIEW OF THE LITERATURE AND REPORT OF SIX CASES

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Much has been published on the cause and treatment of chronic laryngeal stenosis, Lynch [1] giving 278 references in his excellent article. However, there seems to be no unanimity by the various observers in any opinion, except that the condition is one of extreme obstinacy, and that the reestablishment of the normal airway calls into play all the operator's patience and ingenuity. Thomson [2] said that the treatment of stenosis of the larynx is notoriously unsatisfactory.

The sociologic handicap of chronic laryngeal stenosis outweighs the real disability, as there is little danger to life if the patient wears a properly fitted cannula, knows the correct toilet and abstains from strenuous labor, aquatic sports and avocations necessitating presence in a dusty atmosphere.

Fifty years ago, in most cases chronic stenosis of the larynx was considered incurable. Owing to the pioneering of O'Dwyer, Killian, Rogers, Jackson, Lynah, Iglauer, Lynch, Schmiegelow and many others, laryngologists may now say that in most cases the condition is amenable to treatment.

In this article, in an effort to clarify the situation, I have reviewed the literature and given the views of laryngologists treating patients with laryngeal stenosis and also my personal experiences.

Stenosis of the larynx is a narrowing in cross-section of the laryngeal airway. The term chronic is more properly used when the stenosis is cicatrical. When the stenosis is complete, the condition should be termed atresia of the larynx.

Chronic postdiphtheritic stenosis may be of the panic, the spasmodic, and rarely of the paralytic, types; more often it is either of the

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1. Lynch, R. C.: Tr. Am. Acad. Ophth. **33**:355, 1928.

2. Thomson, St. Clair: Diseases of the Nose and Throat, ed. 3, New York, D. Appleton and Company, 1926, p. 618.

hypertrophic or cicatricial form.[3] Only the hypertrophic and cicatricial types are here considered. The exact cause in many cases is obscure. Rogers[4] said: "Hypertrophic subglottic laryngitis is most commonly encountered in retained tube cases, less often in those with a retained tracheal cannula and almost always after diphtheria."

The etiologic factors to be considered are: intubation, tracheotomy, diphtheria and secondary infection, one or more of which may be responsible for the stenosis. According to Rogers,[5] "about one per cent of patients intubated for laryngeal diphtheria are subsequently unable to breathe with an intubation tube or tracheotomy cannula, generally as a result of chronic subglottic laryngitis." Lynah[6] stated that postdiphtheritic laryngotracheal stenosis occurs in about 1 per cent of cases in which intubation or tracheotomy is done, though Cartin[7] reported 350 cases of intubations in none of which the tube was worn chronically. Wilson,[8] citing 352 cases from the Willard Parker Hospital in which intubation was done since September, 1926, said that tracheotomy was necessary in 14 cases because extubation could not be done. In 2 of these, three year tubes were used when one year tubes were indicated. The patients were admitted to the hospital wearing the oversized tubes. Orton[9] reported intubations on 383 persons at the Isolation Hospital in Newark, N. J.; 23 were tracheotomized, and of these, 13 wore the tube constantly.

That stenosis was frequently produced by other causes is evidenced by O'Dwyer's[10] first paper on his method of intubation in which he advised its employment in these cases, and later at the International Congress held in Washington he reported 5 cases in which cure was obtained by this method. Cures were also reported by Lefferts[11] through the use of intubation tubes and his intubation instruments.

Intubational dilatation was satisfactory in the hands of Lynah[12] in the hypertrophic and cicatricial types, but great care had to be exercised not to overdilate the larynx, as a perichondritis might result, adding to the stenosis and making cure more difficult. He added, further,

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3. Jackson, Chevalier: Bronchoscopy and Esophagoscopy, ed. 2, Philadelphia, W. B. Saunders Company, 1927.
4. Rogers, John, and Delavan, D. B.: Tr. Am. Laryng. A. **27**:79, 1905.
5. Rogers, John: Am. J. M. Sc. **135**:575, 1908.
6. Lynah, H. L.: New York M. J. **109**:146 (Jan. 28) 1919.
7. Cartin, H. J.: Intubation of the Larynx, J. A. M. A. **69**:460 (Aug. 11) 1917.
8. Wilson, A. S.: Management of Chronic Tube Cases, read before New York Acad. Med., Nov., 1930.
9. Orton, H. B., in discussion on Wilson.[8]
10. O'Dwyer, J.: New York M. J. **42**:145 (Aug. 8) 1885.
11. Lefferts, George: New York M. Rec. **38**:369 (Oct. 4) 1890.
12. Lynah, H. L.: Laryngoscope **28**:629 (Sept.) 1918.

that he had never seen a properly fitted, undersized intubation tube do any harm, and it is doubtful if they were ever the source of trouble when introduced properly, no matter how often the procedure may be necessary. Clerf [13] stated that in the absence of ulceration it is probable that no injury of the larynx will result from the wearing of a properly fitted intubation tube.

O'Dwyer [10] and many others have held that some of the lesions observed were due to tightly fitting tubes, leaving the tube in too long, to poorly constructed instruments and to injuries resulting from unskilled attempts to intubate.

Wearing an improperly fitted tube, or one too large or with sharp edges at its upper extremity, sets up a reaction about the superior opening of the larynx which is described by Jackson [14] as supraglottic hypertrophy. Also, an intubation tube worn over a long period of time may become encrusted with concretions which produce ulcerations and later cicatrices.

Mallet [15] said: "Prolonged intubation often gives rise to formation of ulcers in the cricoid region, provoking edema below the glottis and laryngeal spasm."

According to Waxham and Witherstine: [16] "The shorter the time the tube is worn, the less likely is one to meet with paralysis of the vocal cords and other conditions."

The so-called noncough-up tube, which often causes pressure necrosis in the subglottic area, is a fruitful source of the stenosis. Iglauer [17] expressed the belief that laryngeal stenosis is usually secondary to pressure necrosis followed by ulceration and subglottic cicatrization. If the intubation tube is too long, it favors this condition. Quoting Fischer: [18] "Recurring laryngeal stenosis is usually caused by forcibly pushing the tube into an edematous or infiltrated mucous membrane. The only safeguard in preventing excessive mechanical injury is to introduce a tube of small caliber." Shurley [19] sharply criticized F. L. Taylor's recommendation to use a tube in number next above the age of the child.

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13. Clerf, L. H.: Laryngoscope **36**:912 (Dec.) 1926.

14. Jackson, Chevalier: Peroral Endoscopy and Laryngeal Surgery, Philadelphia, W. B. Saunders Company, 1915.

15. Mallet, H.: Rev. méd. de la Suisse Rom. **35**:465 (Aug.) 1915.

16. Waxham and Witherstine: Intubation of the Larynx, in Sajous: Annual and Analytic Cyclopedia of Practical Medicine, ed. 10, Philadelphia, F. A. Davis Company, 1927, vol. 5, p. 807.

17. Iglauer, Samuel: Personal communication, Nov. 24, 1930.

18. Fischer, Louis: New York M. J. **88**:193, 1908.

19. Shurley, B. R.: Prolonged Intubation Tubes, J. A. M. A. **41**:80 (July 11) 1903.

Tracheotomy in which the laryngeal cartilages are injured, however, is the most frequent cause of chronic laryngeal stenosis, according to Jackson;[3] indeed, 75 per cent[20] of his cases were due to so-called high tracheotomy wherein the cricoid cartilage was severed, this being the commonest error. Stabbing the cricothyroid membrane and hacking of the tracheal cartilages in hurried tracheotomies are often determining factors. Thomson,[21] after having treated many of these patients, said: "The usual operative error is to divide the first ring of the trachea or even the cricoid and so introduce the cannula into the narrow, vascular, glandular and sensitive subglottic larynx and not in the more spacious trachea. This gives rise to irritation and hypertrophy." Quoting Schmieden[22] on tracheotomy: "We absolutely condemn division of the cricoid cartilage or crico-thyroid membrane, because it destroys the basis of the laryngeal apparatus. We hold that the old cricotracheotomy should be reserved for very exceptional conditions." Negus,[23] in his monumental work on the larynx, said: "The cricoid is particularly important in keeping the air-way patent and if it is fractured serious obstruction to respiration is produced." New[24] stated that in his experience the stenosis is usually due to high tracheotomy. Clerf[25] also concurred in the belief that high tracheotomy was unquestionably the cause of several cases. Lierle[26] attributed practically all of his cases to faulty tracheotomy or intubation. Stucky,[27] in reporting 5 cases, said that 3 were undoubtedly due to faulty tracheotomy or intubation. Woody[28] also believed that in his experience stenosis was most frequently caused by faulty intubation or tracheotomy. Sonnenschein[29] expressed the opinion that the condition was usually due to some error in intubation or tracheotomy or often to prolonged retention of the intubation tube.

Even though the cricoid is not severed, if the cannula is placed high and in contact with the cricoid, a perichondritis may ensue. Wearing a cannula of improper size or shape, carelessness in the after-care or neglect of ordinary cleanliness in the wearing of the cannula are other causes of perichondritis and subsequent stenosis.

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20. Jackson, Chevalier: Surg., Gynec. & Obst. **32**:392 (May) 1921.
21. Thomson, St. Clair: J. Laryng. & Otol. **46**:817 (Dec.) 1931.
22. Schmieden, V.: Course of Operative Surgery, translated by A. Turnbull, New York, William Wood & Company, 1912.
23. Negus, V. E.: The Mechanism of the Larynx, St. Louis, C. V. Mosby Company, 1929.
24. New, G. B.: Personal communication, Nov. 29, 1930.
25. Clerf, L. H.: Personal communication, Dec. 3, 1930.
26. Lierle, D. M.: Personal communication, Dec. 2, 1930.
27. Stucky, J. A.: Personal communication, Nov. 26, 1930.
28. Woody, S. S.: Personal communication, Feb. 17, 1931.
29. Sonnenschein, Robert: Personal communication, Dec. 8, 1930.

Lynah [12] expressed the belief that ulcerations from tracheal cannulas are rare, even with the old style fenestrated type, but Hitschler [30] stated that "occasionally granulations are found at the external orifice and in the trachea itself, the latter being especially the case when a fenestrated tube is employed."

Anterior and posterior spur formations are frequent, especially when an oversized or improperly curved cannula is worn in which the sharp edges of the lower end impinge against the larynx or trachea, rubbing and irritating the walls with each respiratory movement.

Thost [31] expressed the opinion that too small a tracheal incision or too large a cannula are also factors in stenosis following tracheotomy, because of distortion of the trachea and subsequent trauma due to displacing the cartilaginous rings and mucosa.

Another contributing factor is trauma due to the careless introduction of the cannula without using the pilot, or Trousseau dilator.

Diphtheria itself in the absence of intubation or tracheotomy may cause stenosis, as the disease is essentially a necrotic process and the subsequent changes that occur in the larynx and trachea depend on the duration of the disease and the degree of involvement. Coplin,[32] in his "Manual of Pathology," reported that of 220 patients examined by Councilmen the larynx was involved more than any other part of the airway. Quoting Clerf: [13] "In severe laryngeal cases, necrotic changes occur not only in the submucosa but also in contiguous structures, so that perichondritis with considerable cartilage loss may ensue. Cicatricial stenosis may follow during the stage of resolution and healing. With this, mixed infection is the rule so that we may concurrently find streptococcus, staphylococcus, pneumococcus and other bacteria." There may result excessive hypertrophy about the upper part of the larynx, resembling an extensive edematous condition, or there may be marked swelling of the subglottic tissues. The latter condition is commonly bilateral, involving the tissues forming the conus elasticus and encroaching on the lumen on each side, resembling in appearance hypertrophied turbinates.

Jackson [3] called attention to the fact that the disease for which the intubation was indicated produced stenosis even when no intubation was done, and also that in cases of prolonged intubation diphtheria was in part probably responsible.

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30. Hitschler, W. A.: Diseases of the Larynx, in Sajous: Annual and Analytic Cyclopedia of Practical Medicine, ed. 10, Philadelphia, F. A. Davis Company, 1927, vol. 6, p. 304.

31. Thost, A.: Die Veränderungen der obern Luftwege nach dem Luftröhrenschnitt und deren Behandlung, Weisbaden, J. F. Bergmann, 1911.

32. Coplin, W. M. L.: Manual of Pathology Including Bacteriology, ed. 4, Philadelphia, P. Blakiston's Son & Co., 1905, p. 684.

Lynah [12] reported the cases of ten patients who responded so readily to antitoxin that intubation was not necessary. All these patients, however, became dyspneic in from seven to ten days after the primary diphtheritic process had subsided and required intubation to relieve the stenotic lesion. Direct laryngoscopy revealed subglottic edema.

Johnson [33] expressed the belief that the exact cause producing the condition is obscure in the majority of cases, and that the acute laryngeal infection itself frequently may cause ulceration which terminates in scar formation and laryngeal stricture. In his group of 12 cases there were no high tracheotomies, and none showed any injury to the cricoid. Imperatori [34] reported a case of tracheal stenosis following diphtheria in which he stated that the diphtheritic erosion and repeated intubations caused the cicatricial stenosis. Tucker [35] mentioned the same factor as well as trauma in attempting intubation, as a possible cause of injury to the arytenoids. Arbuckle [36] stated that the most frequent cause of necrosis of the laryngeal tissues is diphtheria and the accompanying mixed infection. Quoting Lewis,[37] who has seen a number of these cases: "Our experience was that the majority was the result of the primary infection producing a perichondritis, and some were due to faulty tracheotomy." Myerson [38] gave as the second most important cause, "the diphtheritic infection itself, causing ulceration and necrosis with resulting cicatrization and stenosis." Lynch [39] also believed that the diphtheritic process itself causes stenosis in some cases.

Secondary infection (in the absence of intubation or tracheotomy) is a minor factor, according to Myerson.[38] It is difficult to arrive at a positive conclusion regarding its importance. McGinnis [40] reported two cases which may have been due to secondary infection; however, the obstruction was not enough to be of serious import.

The lesion responsible for the stenosis may be supraglottic, subglottic or a combination of both. However, the fundamental structural defect is cicatricial tissue. When supraglottic, the base of the epiglottis, the glosso-epiglottic fold, the arytenoids or the ventricular bands are usually involved. It may be ulceration, hypertrophy, papillosis or edema, or any combination of the preceding, with subsequent formation of webs

33. Johnson, L. F.: Laryngeal Stenosis, read before Newton Med. Club, Boston, Dec., 1930.

34. Imperatori, C. J.: Cicatricial Stenosis of the Trachea, Arch. Otolaryng. **1**:624 (June) 1925.

35. Tucker, Gabriel: Laryngostomy for Laryngeal Stenosis, Arch. Otolaryng. **1**:157 (Feb.) 1925.

36. Arbuckle, M. F.: Ann. Otol., Rhin. & Laryng. **39**:134 (March) 1930.

37. Lewis, F. O.: Personal communication, Nov. 26, 1930.

38. Myerson, M. C.: Personal communication, March 31, 1931.

39. Lynch, R. C.: Personal communication, Nov. 24, 1930.

40. McGinnis, Edwin: Personal communication, Nov. 22, 1930.

and cicatrices. The aryepiglottic folds and the arytenoids may also swell enormously. This may be caused by the diphtheritic infection itself or by the head of the intubation tube being too large or improperly shaped.

The ventricular bands are usually seen to be greatly thickened and edematous, so that when the tube is removed the glottis is immediately occluded. With each attempt at inspiration they are drawn inward, increasing the respiratory difficulty. Although the redundant tissue obstructs the view in the larynx, it offers but little resistance to instrumentation when the glottis is entered, and there is no gripping of the bronchoscope or dilator on withdrawal.

The subglottic type involves the region of the cricoid, and the tissues forming the conus elasticus where the loosely attached submucosa is richly supplied with blood and lymph vessels. This arrangement is particularly adapted to deep infiltration, giving rise to an obstructive chronic hyperplasia which may be annular or lateral, more or less symmetrical masses on each side, immediately below and occasionally integral with the cords. Here the lesion may consist of symmetrical turbinal-like swellings encroaching on the lumen from either or both sides, papillomatous masses, ulceration and inflammation, perichondritis, chondritis, webs, cicatrices or actual loss of cartilage with partial or complete atresia. A heavy mass of cicatricial tissue may have resulted from the perichondritis, with consequent narrowing of the airway on contraction of the same. Where the cartilage has been destroyed with resultant collapse of the framework which holds the larynx open, the airway is practically obliterated, and the most severe and refractory types of cicatricial stenosis ensue.

All these patients present themselves wearing an intubation tube or tracheal cannula, with a history of being unable to abandon them permanently, or of having worn an intubation tube or cannula some time previously, and of gradual development of difficulty in breathing. The diagnosis is usually self-evident. However, careful study of the case is necessary to ascertain the exact location of the stenosis and to outline a method of treatment. Inspection should be made with a mirror, by direct laryngoscopy and, if necessary, a small bronchoscope may be used. Occasionally retrograde laryngoscopy may be required.

Roentgenograms [41] and fluoroscopic observations are often needed, as the extent of the stenosis can be well demonstrated by these methods. Radiopaque oils can be instilled into the larynx in the irregular subglottic types to aid in visualization when tracheotomy has been performed.

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41. Pancoast, H. K.: Roentgenology of the Upper Respiratory Tract, J. A. M. A. **95**:1318 (Nov. 1) 1930.

Hypertrophied or infected tonsils and adenoids should be removed as a preliminary procedure to any attempt to reestablish a normal physiologic airway.

Treatment of the different types of laryngeal stenosis varies with the pathologic process present. Radical procedures should be postponed as long as possible. Paunz [42] said that it is inadvisable to perform laryngofissure on children under the age of 4 years. Runge [43] recommended, in very young children who for various reasons are wearing a tracheal cannula that cannot be safely abandoned, awaiting the growth of the larynx and trachea instead of proceeding with operative intervention. Some thought that children would not stand the operation well, although Jackson [44] said that the operation itself is relatively a minor part, painstaking after-care being essential to success. Others were under the impression that laryngofissure would interfere with the development of the laryngeal cartilages. Arbuckle,[36] however, reported that in so far as he has been able to observe from time to time since operation, the larynx is keeping step with other parts of the body.

In the supraglottic type in which the stenosis consists of a simple hypertrophy or edema, the use of a smaller and correctly formed intubation tube may be all that is required. In hypertrophy, accompanied by papillomatous tissue or hyperplasia, galvanocautery may suffice, though endoscopic excision with a biting forceps is preferable, the intubation tube being replaced if this is not a factor in causing the stenosis. Webs may be incised or divided with the galvanocautery under direct laryngoscopy.

In general, however, it may be said that a tracheotomy should precede these operations, as the irritation of the intubation tube is avoided, time is afforded for proper study of the condition and the patient is far safer with the tracheal cannula. If the cannula is of the proper size and shape (preferably the Jackson type), and the tracheotomy is an orderly procedure, placed exactly in the midline and well below the cricoid, the larynx is put at rest, and ultmiate cure with a patent airway and well functioning cords is facilitated. If the laryngeal framework has not collapsed and if an opening is present in the larynx for a potential airway, dilation with Jackson's dilators should be the next step, beginning with the smaller ones and gradually increasing the size, as the lumen of the larynx is enlarged. The triangular type is used for lesions limited to the larynx, and if the trachea is involved the round dilators are also required. The dilators may be lubricated with heavy

42. Paunz, Mark: Ztschr. f. Hals-, Nasen- u. Ohrenh. **22**:424, 1929.

43. Runge: Ztschr. f. Hals-, Nasen- u. Ohrenh. **24**:576, 1929.

44. Jackson, Chevalier and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929.

mineral oil, avoiding heavy petrolatum or jellies which cause distress if lodged below the tracheotomy. A clean cannula, taped and containing the pilot, must be previously prepared. When ready to introduce the dilator into the larynx, an assistant removes the cannula. On withdrawing the dilator, the clean cannula is quickly inserted. Dilation may be repeated at weekly intervals.

In the subglottic cases, tracheotomy should be the preliminary procedure where it has not already been done. If the stenosis is due to high tracheotomy, a low tracheotomy must be the first step and, if possible, two or three intact tracheal rings should be left between the old and the new openings. When a second tracheotomy is done, time should be allowed for the tissues to reunite about the new wound and for the old fistula to close before commencing dilation.

The peroral method of dilation is applicable in all cases in which there is not a complete atresia of the airway or too great a loss of the laryngeal cartilages. It should be given a thorough trial before the more radical external methods are employed. Care must be exercised that overdilation does not cause perichondritis, and if an acute laryngotracheitis develops, dilation should be discontinued until the inflammation subsides.

An important adjunct, particularly in children, to the dilation is the gradual closure of the tracheal cannula with grooved rubber stoppers, as suggested by Jackson,[3] because the normal airway is enlarged through exercise of the muscles and enforced laryngeal function. Closure, however, should not be made if much expectoration is present, or if there is a mucopurulent discharge from the cannula. Numerous observers report cures by this method.[45] Johnson [45a] reported electrically heated bougies similarly employed as giving excellent reults in the more refractory cases.

The use of an expansile rubber tube was first proposed by Killian in connection with laryngostomy, and has been used since then without laryngostomy with excellent results.

Iglauer [46] described an ingenious and original method of continuous dilation which seems entirely logical. A stout silk cord attached to an eyed probe is passed through the tracheal fistula from below until it presents itself in the oropharynx. The cord is then pulled out of the mouth, leaving one end hanging from the tracheal opening. A piece of pure soft rubber tubing which has been doubled on itself is then attached to the oral end of the cord and pulled into the larynx. The tracheotomic

45. (*a*) Johnston, W. H.: Ann. Otol., Rhin. & Laryng. **38**:1145 (Dec.) 1929. (*b*) Lederer, F. L.: Personal communication, Nov. 22, 1930. (*c*) Footnotes 8, 9, 24, 33, 36 and 44.

46. Iglauer, Samuel: Ann. Otol., Rhin. & Laryng. **27**:1233 (Dec.) 1918.

end of the cord is wound around the cannula to retain the tubing in place. This method appears far more preferable than prolonged intubation with metal or hard rubber tubes, as the soft rubber tubing will not cause decubitus ulcers, readily adapts itself to the lumen of the larynx, and by gentle pressure promotes resorption of the cicatricial tissue.

Tucker [47] obtained most satisfactory results from a modification of this method, wherein a plug made from soft rubber over a copper wire core is introduced retrograde and maintained in position by the tracheal cannula. In both of these methods a real or potential passage through the larynx to the tracheal fistula is predicated.

When complete atresia of the larynx exists and intralaryngeal operative procedures with peroral methods of dilatation have not been successful in reestablishing a physiologic airway, an external operation is necessary. Of the external operative methods of dilatation, Schmiegelow's [48] technic is the less radical and has given excellent results in 18 cases. Following a low tracheotomy he performs a thyrotomy, completely exposing the larynx above the tracheotomy. Scar tissue is excised, and a thick-walled rubber tube is laid in the laryngeal cavity extending upward from the tracheal cannula to the level of the arytenoids. The tube is anchored in place by a silver wire passed through the alae of the thyroid cartilages and the walls of the tube. Then the silver wire is cut off at each side flush with the skin surface and the skin drawn aside so that the ends of the wire are covered. The tube is left in situ from three weeks to three months and then withdrawn through the mouth, the silver wire bending as traction is made on the tube and coming out with it.

Laryngostomy, as suggested by Killian,[49] Melzi and Cagnola,[50] Sargnon and Barlatier,[51] Thost and Moure,[52] should be the operation of last resort and is indicated only when all other methods fail.

The modification as described by Jackson [44] has been evolved from much experience, and, if painstakingly followed, permanent relief of the stenosis is usually assured.

The obstructive cicatricial tissue is dissected out and the larynx is kept open for a long period, so that the newly formed lumen becomes

47. Tucker, Gabriel: Apparatus for Treatment of Laryngeal Stenosis, Tr. Sect. Laryng., Otol. & Rhin., A. M. A., 1930, p. 213.

48. Schmiegelow, E.: Stenosis of the Larynx, Arch. Otolaryng. **9**:473 (May) 1929.

49. Killian, R., cited by Knick, A.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **10**: 307 (Nov. 8) 1924.

50. Melzi, U., and Cagnola, A.: Ann. d. mal. de l'oreille du larynx **34**:536, 1909.

51. Sargnon, A., and Barlatier, R.: Le traitement chirurgical des sténoses laryngo-trachéales, Paris, A. Maloine, 1910.

52. Moure and Richard: Ann. d. mal. de l'oreille du larynx **39**:193, 1913.

covered with epidermal epithelium. A dilator of some type, rubber, gauze or sponge,[27] is constantly kept in the trough during the period of treatment. If sufficient cartilaginous framework remains to give support to the airway, a cure can be obtained. If the cartilaginous framework has collapsed, it may be restored by transplantation of bone and cartilage, but the difficulties are great. Nassau's [53] method of plastic closure following laryngostomy seems the one of choice as it has given excellent results in his series. It is presumed, of course, that this treatment is carried out in a well regulated hospital where the patient is at all times under careful supervision of competent and watchful nurses.

REPORT OF CASES

The following case reports, although a small series, are of interest in that they represent both supraglottic and subglottic lesions, that the etiology varied, and that conservative and radical methods were necessary to reestablish the physiologic airway.

CASE 1.—C. B., a Negro boy, aged 18 months, was admitted to the isolation department of the Louisville City Hospital on June 17, 1929, with a diagnosis of "laryngeal diphtheria." Because of the severe respiratory embarrassment, intubation was done immediately. After he had repeatedly coughed up the tube, tracheotomy was done on the following day, with prompt cessation of respiratory distress. Beginning on July 10, numerous attempts were made at decannulation, all of which were unsuccessful.

I first saw the child on October 20, at which time direct and retrograde laryngoscopy revealed supraglottic edema and marked infiltration of the conus elasticus. Medicated vapors from a croup kettle were employed for several days, and the edema subsided. Dilation was then begun with the Jackson type dilators, being done at weekly intervals, and the cannula partly closed with grooved rubber stoppers. The airway was functioning so well that by December 25 it was possible to occlude about three fourths of the lumen of the cannula, when the child developed a fever and sore throat. Inspection revealed a few inflamed areas and eruptions on the fauces. This was followed in several days by papules and vesicles over the body, and was diagnosed by the pediatric staff as chicken-pox. Dilation was discontinued during the acute infection.

On December 31, I was advised of the patient's death.

Autopsy, which was performed with permission, revealed only a few congested stria in the muscular wall of the left auricle. There were no gross pathologic lesions in the other vital organs.

The nurses on duty at the time of the child's death reported that he was bright and cheerful and playing about as usual, when he lay on the floor apparently "shamming" sleep, as was his habit. About half an hour later it was discovered that he had died.

I am inclined to view the cause of this patient's death as due to asphyxia and not to any cardiac involvement.

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53. Nassau, C. F.: Closure of Laryngostomic Fistulas, Arch. Otolaryng. **1**:632 (June) 1925.

Zinn [54] reported a case in which the child was progressing nicely with a similar outcome.

Case 2.—W. F., a white girl, aged 4 years, was admitted to the isolation department on June 30, 1930, with a diagnosis of "laryngeal diphtheria." As the child was very cyanotic and dyspneic, intubation was performed promptly. Extubation was attempted on July 12, but the ensuing respiratory distress was so pronounced that the tube was reintroduced. A low tracheotomy was then done.

Direct laryngoscopy revealed a large ulcer at the base of the epiglottis with erosion and edema of the anterior half of the ventricular bands. After two weeks, the cannula was partly occluded. Direct inspection revealed that the ulcers had healed, but as there seemed to be some narrowing of the subglottic space, dilation was instituted. Four weeks later the cannula was entirely closed, and after a test period of one week decannulation was performed.

On Sept. 14, 1930, the patient was discharged as cured, the tracheotomy fistula having closed, and there was no respiratory distress. The voice is of normal quality.

Case 3.—R. N., a white girl, aged 4 years, was admitted to the isolation department on Nov. 24, 1929, with a diagnosis of "laryngeal diphtheria." Intubation was necessary because of the extreme cyanosis and dyspnea. On December 2 the membrane peeled from the throat. The following day there was great inspiratory difficulty with marked indrawing of the suprasternal fossa, intercostal spaces and epigastrium. Asphyxiation seemed imminent and a hurried tracheotomy was done, with prompt cessation of the symptoms.

I saw this child on December 31, at which time a direct laryngoscopy and insertion of a small bronchoscope showed that the cricoid cartilage had been severed. On January 22 a low tracheotomy was performed, leaving two intact tracheal rings between the old and new tracheal incisions. On February 22, tonsillectomy and adenoidectomy were done, as suggested by Orton,[55] because the child had a chronic ethmoiditis and the tonsils were much enlarged. The ethmoiditis cleared up with little treatment, after which dilation and partial occlusion of the cannula were instituted. However, on all attempts for complete occlusion of the cannula, considerable respiratory distress ensued. A soft rubber tube was then prepared, after the method as suggested by Iglauer and anchored in the larynx. This was permitted to remain in situ for two weeks, when it was withdrawn and a larger tube substituted. In three weeks this was removed, and on corking of the cannula no respiratory distress was encountered. The stenosis responded to these procedures, and after a test period of six weeks the cannula was withdrawn and the tracheotomy fistula permitted to close.

On July 24, the patient was discharged as cured. In November, 1930, following an attack of measles she had a slight recurrence of respiratory stridor. A croup tent gave her relief, and after recovery from the measles dilation was resumed for five weeks, when she was again discharged as cured. Since that time there has been no recurrence of the stenosis. Her voice is somewhat husky and rough.

Case 4.—M. L., a Negro girl, aged 2 years, was admitted to the isolation department on June 6, 1930, with a diagnosis of "laryngeal diphtheria." She had been ill for one week previous to admission with a sore throat, called tonsillitis. She was immediately given 20,000 units of antitoxin intravenously. The following

54. Zinn, W. F.: Personal communication, May 2, 1931.

55. Orton, H. B.: Postdiphtheritic Stenosis of the Larynx, Arch. Otolaryng. **3**:521 (June) 1926.

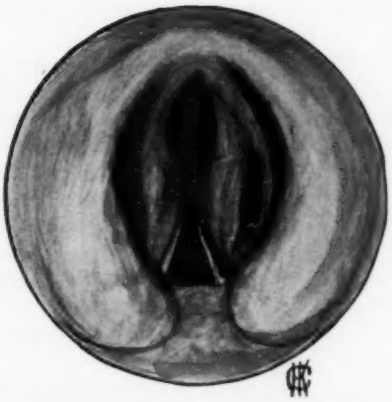

Fig. 1 (case 1).—Direct laryngoscopic image showing supraglottic edema.

Fig. 2 (case 3).—Direct laryngoscopic image showing subglottic edema and stenosis due to severance of the cricoid.

Fig. 3 (case 4).—Direct laryngoscopic image showing the ulcer on the left anterior aryepiglottic fold and the right ventricular band; subglottic edema.

day severe dyspnea developed and preparations were made to do intubation under direct laryngoscopy, when the larynx was seen to be intensely congested and swollen with several large denuded areas in the aditus. Because of the ulceration, it was decided tracheotomy would be the wiser procedure. Four weeks later laryngoscopic examination revealed that the larynx was healed but that several webs partially closed the anterior glottis. These were incised under direct inspection and dilation was begun. After three weeks the cannula was partly closed with grooved stoppers. Dilation was also continued, and on Jan. 4, 1931, it was possible to close the cannula entirely. After a test period of one month, the cannula was withdrawn, and the fistula allowed to close.

The child was discharged on February 16, and there has been no recurrence up to the time this paper was written.

Case 5.—P. M., a white boy, aged 6 years, was admitted to the Louisville City Hospital on Sept. 12, 1930, having considerable difficulty in breathing. There was a previous history of diphtheria.

Fig. 4 (case 6).—Direct laryngoscopic image showing nodules and webs.

Direct laryngoscopic examination revealed a large, thick, fibrous band connecting both arytenoids and extending over the glottis. This was severed, and the Jackson type triangular dilators were used at biweekly intervals for five weeks.

The patient was discharged on October 20 as cured. The voice was normal.

Case 6.—J. C., a white boy, aged 5 years, had been in the Louisville City Hospital for seven months following "laryngeal diphtheria." A low tracheotomy, correctly placed, had been done.

I was asked to see this child on Jan. 14, 1930. Direct laryngoscopy revealed the aditus of the larynx filled with papilloid nodules and considerable distortion of the ventricular bands due to cicatricial webs. The nodules were removed with a punch forcep and the webs incised. It was impossible, however, to penetrate the glottis with the smallest size dilator. A filiform bougie was introduced through the tracheal fistula, and after considerable difficulty the glottis was entered from below. Treatment was then halted during my absence from the city. During this period, fenestrated cannulas were substituted for the Jackson type. The child also developed measles. Two months later it was impossible to enter the glottis from either direction with the smallest size bougie. As the child was such a poor risk, it was decided to delay radical operative methods until a later date.

On March 3, 1931, a small bronchoscope was introduced between the ventricular bands, and the glottis was seen to be closed by a dense cicatrix. A special handle carrying a no. 15 Bard-Parker blade was then devised, and the cicatrix was divided in the midline under direct inspection. It was then seen that there was also complete atresia of the subglottic space. Roentgenologic studies revealed that the stenosis extended down to the upper margin of the tracheotomy. Thyrotomy was then done, the scar tissue dissected out and a soft rubber tube laid in the larynx, the upper end of the tube projecting just beyond the glottis and the lower end in contact with the tracheal cannula. The wound was closed with interrupted sutures through the muscles and skin. A firm stay suture was introduced just above the cannula, and the cord was attached to the lower end of the rubber tubing wound

Fig. 5 (case 6).—The dark area between the light sections, outlined with dotted lines, shows a portion of the larynx with complete atresia.

around the cannula. This method of continuous dilation has been carried out for five weeks, and it is felt that it will be successful in reestablishing a patent physiologic airway.

CONCLUSIONS

1. To no one cause can postdiphtheritic stenosis of the larynx be ascribed. Errors in intubation or tracheotomy, prolonged retention of tubes and diphtheritic infection are the chief causes, in the order named.

2. Intubation by direct laryngoscopy is to be preferred, because it can be readily learned, trauma to the larynx is lessened, and at the time of intubation the larynx can be studied.

3. Tubes should not be worn over two weeks; probably a shorter period is preferable. Tracheotomy is indicated if extubation is impossible at this time.

4. Dilation should be instituted early if decannulation is impossible, as the cicatrix is still soft and responds to dilation more readily.

5. Jackson's method of dilation and occlusion is the procedure of choice wherever applicable. When continuous dilation is necessary or desirable, the method of Iglauer seems most logical.

6. Knowledge of the nature and extent of the stenosed area, which can be determined by direct and retrograde laryngoscopy and by roentgenologic studies, is necessary before treatment is instituted.

7. As most of the cases of laryngeal stenosis follow diphtheria in children of preschool age, early immunization of the very young should be urged.

8. The prognosis for ultimate functional recovery is favorable if the cartilaginous framework is not entirely destroyed.

9. Treatment should be carried out in a well regulated hospital where the patient is under the constant supervision of watchful nurses especially trained for this work.

VARIATIONS IN THE TEMPERATURE OF THE MUCOUS MEMBRANE OF THE NOSE

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Much has been written concerning the changes in the temperature of the extremities due to sympathetic vasoconstrictor activity. Few references are found in the literature regarding variations in the temperature of the nasal mucous membrane.

For several years, during the routine preoperative examination of patients before operations on the thyroid, I have noted that the mucous membrane of the nose in patients with high basal metabolic rates has been thin. Edema, except in the presence of an acute upper respiratory infection, has been rare. On the other hand, in patients with metabolic rates below normal, the mucosa has frequently been boggy and edematous, much the same as the nasal membrane of an allergic patient. This clinical observation suggested investigation of possible temperature changes in the nasal membrane similar to the changes in the extremities in Raynaud's and Buerger's disease. For this work I have used the same type of instrument, a dermatherm, as used by the neurologist and neurosurgeon in studying vasoconstrictor activity and changes in the temperature of the extremities.

No instrument in use provided a thermocouple that permitted contact with the nasal membrane. Through the cooperation of the Tycos Instrument Company, I have obtained a special applicator with contact points on the side. This applicator is small enough to permit easy introduction into the nose so that contact can be made with the mucosa of the inferior turbinate.

TECHNIC

The technic consists in having the patient hold his breath during the contact period. This period requires only ten seconds and prevents variation in the readings due to inspiration and expiration. Moderate pressure against the mucous membrane is used. An assistant makes the galvanometer reading and times the contact period.

It is only in highly nervous persons and in those with mechanical obstruction that any difficulty is encountered. No anesthesia is used on the nasal membrane, as such a procedure would affect the readings. The applicator is sterilized between each case by soaking in 5 per cent phenol solution.

Repeated observations have been made in individual cases as a check on the reliability of the findings. The chief difficulty has been in making the same pressure of the applicator on the nasal membrane.

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OBSERVATIONS

I am reporting the findings in a group of seventy-five cases. The patients have been roughly divided into five groups:

(*A*) Those with no demonstrable infection of the nasal membranes or sinuses.

(*B*) Those with an acute or subsiding infection.

(*C*) Those with high metabolic rates.

(*D*) Those with low basal metabolic rates.

(*E*) Those with nasal allergy.

The normal temperature of the inferior turbinate has been found to be 32 C. (89.6 F.). Patients with acute nasal involvement and with high basal rates have had higher temperatures. Those with allergy and those with low basal rates have had lower temperatures. The averages in the five groups have been recorded as differences between the nasal and the oral temperature. Thus the normal nasal temperature of the inferior turbinate, which is 32 C., is recorded as —5 C. (—9 F.). This of course, is in a patient with a normal oral temperature of 37 C. (98.6 F.). The nasal temperature reading has been subtracted from the oral temperature, which is taken at the same time.

Normal	—5.0 C. (—9.0 F.)	32.0 C. (89.6 F.)
Acute infection	—3.2 C. (—5.8 F.)	33.8 C. (92.8 F.)
Hyperthyroidism	—4.5 C. (—8.1 F.)	32.5 C. (90.5 F.)
Hypothyroidism	—6.6 C. (—11.9 F.)	30.4 C. (86.7 F.)
Allergic	—5.9 C. (—10.6 F.)	31.1 C. (87.9 F.)

The possible variants in such a study as this are many. Therefore, all cases have been carefully selected, special care being taken to check the basal tests and laboratory work, which included a study of nasal smears for pus cells, eosinophils, etc.

Many persons are seen with unilateral edema or with an infectious process limited to one side of the nose. In these cases there is a difference in the temperature of the two sides. In patients with unilateral edema, the temperature was lower on the edematous side. In the cases of unilateral infection, the temperature was higher on the affected side. Some difficulty has been encountered in evaluating the findings in cases of infection. A patient with maxillary sinusitis on one side only showed an increase in the nasal temperature. In checking the opposite side with a clear maxillary sinus, there was occasionally, also, an increased temperature. Further study in such cases usually revealed an ethmoid involvement, which would account for the change. Many more cases must be studied before the true value of the findings in cases of infection can be demonstrated.

Krukower [1] reported variations in the temperature of the nose as recorded with glass thermometers. He stated:

The body temperature plays some part in the nasal temperature; the temperature of the nose varying directly as that of the body. Outside temperature has no marked influence.

In cases in which there was every indication of disease of the sinuses but the nasal temperature was not elevated, 5 cc. of milk was injected, and the temperature of the unaffected side of the nose returned to normal in two or three days, while the temperature of the diseased side remained elevated

Special applicator for obtaining the temperature of the mucous membrane of the nose.

His conclusions are as follows:

1. In maxillary sinusitis the temperature in both meatuses is elevated.

2. In unilateral maxillary sinusitis the diseased side has a higher temperature than the unaffected side.

3. Long-standing, chronic disease of the sinuses causes no notable rise in temperature, but

4. An injection of 5 cc. of milk causes a continual rise in temperature in these cases.

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1. Krukower, I. M.: Veränderungen der Temperatur in der Nasen als Basis für die diagnostisches Verfahren, Monatschr. f. Ohrenh. **63**:838 (Sept.) 1929.

My findings agree with the foregoing, except that I have not used the injection of milk to demonstrate changes in temperature in long-standing cases.

The most interesting group of cases, those with variations in the basal metabolic rates from —30 to +52, have shown the most constant variations, ranging from a nasal temperature of 30.2 C. (86.3 F.) in the patient with a basal rate of —30 to a temperature of 32.5 C. (90.5 F.) in a patient with a basal rate of +52 and a temperature of 33.2 C. (91.7 F.) in a patient with a +36 basal rate.

This change in temperature of the nasal mucosa has been so constant that I think that there is direct nasal evidence of stimulation of the sympathetic nervous system in cases of thyroid toxicity. The similar findings between allergic patients and hypothyroid cases is interesting. Further study is being carried out especially in these two groups. Several patients with vasomotor rhinitis during the menopause have also shown the same findings (low nasal temperature). A careful allergic check-up on these persons has revealed nothing. The only explanation of the low nasal temperature must be associated with the general circulatory changes that are present.

Reports of interesting studies of changes in temperature are found in the literature in increasing numbers. There are numerous other reports regarding the effects of vasoconstriction.

The late Gustav Alexander [2] stated that the "rotary vertigo of neurasthenics is caused by vasomotor disturbances, first of all by vasospastic conditions in the vascular regions of all the apparatuses ruling over static."

This, according to Bayliss' [3a] conclusions, would have to be caused by the action of the sympathetic nervous system.

Morton and Scott [4] reported the use of spinal anesthesia as a means of estimating the participation of the lumbar sympathetic nervous system. "This is applied as a functional test for vascular disease of the lower extremities following spinal anaesthesis. It is also used as a functional test for Hirschprung's disease. The motility and expulsive power of the large bowel is determined with a barium enema."

Adson [5] reported the application of sympathectomy in Raynaud's and Buerger's disease and in selected cases of hypertension and arthritis.

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2. Alexander, Gustav: Anatomy and Clinical Importance of Ear-Findings in the Neurological Symptom-Complex, Proc. Inter-State Post-Grad. M. Assemb., North America **3**:401, 1927.

3. Bayliss, W. M.: (*a*) The Vasomotor System, New York, Longmans, Green & Co., 1923, p. 34; (*b*) p. 45.

4. Morton, J. J., and Scott, W. J. M.: Studies on the Activity of the Lumbar Sympathetic Nervous System, Ann. Surg. **92**:919 (Nov.) 1930.

5. Adson, A. W.: Results of Sympathectomy in the Treatment of Raynaud's and Buerger's Disease, Proc. Inter-State Post-Grad. M. Assemb., North America **3**:386, 1928.

He stated, in regard to the arthritic case, that "The result was most dramatic. It is an investigative case and we are not advising it for all arthritics."

Bayliss [3b] stated that "the effects of a change in blood supply of an organ, so far as they are readily detected by brief observation, are changes in the depth of the red color, in the rate of overflow from the veins, in the size of the organs, and, in some cases, in its temperature."

Regarding temperature, he stated that "In the case of the skin which is normally at a lower temperature than the blood, an arterial dilatation produces a rise in temperature. On the other hand, a capillary dilatation does not of necessity raise the temperature, although there is hyperemia."

He further stated, "Since we have seen all the vasoconstrictors arise from the sympathetic and from no other source, we should not expect to find any vasodilators of sympathetic origin."

CONCLUSIONS

1. The temperature of the mucous membrane of the nose (inferior turbinate) is altered (*a*) in patients with infection, (*b*) in those with abnormal basal metabolic rates, (*c*) in those with vasomotor rhinitis and (*d*) in those with allergy.

2. The changes in temperature in patients with abnormal basal metabolic rates, vasomotor rhinitis and allergy may be due to the variations in the sympathetic vasoconstrictor activity, as evidenced by the changes in color, volume and temperature of the turbinates.

PRIMARY JUGULAR BULB THROMBOSIS

A STUDY OF TWENTY CASES

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NEW YORK

The difficulty encountered in the recognition of a general infection of otitic origin has always been considerable, the main reason being the large number of diseases which may be simulated and the few symptoms which are characteristic of this condition. This difficulty is much increased in cases of general infection of unknown origin in which the aural examination reveals findings ordinarily regarded as unimportant.

In the study of our twenty cases of primary jugular bulb thrombosis we were so impressed with the striking similarity of the aural manifestations, clinical course and operative findings that we were led to the opinion that this form of sinus thrombosis represents a clinical entity. Because of an apparently unimportant, vague or indefinite aural history in cases of primary jugular bulb thrombosis, the condition is more likely to be seen at the onset by the internist or by the pediatrician than by the otologist. For similar reasons, these cases are usually seen less frequently in special hospitals. There are still some otologists who question the existence of the condition to be described. The importance of early diagnosis becomes evident when one realizes that in order to obtain a favorable outcome of this serious illness, prompt surgical intervention is imperative.

The term primary jugular bulb thrombosis is employed to designate the formation of an infected thrombus in the dome of the jugular bulb. With the advance of the disease, the thrombosis extends to the sigmoid sinus, and may even reach the lateral sinus. The mastoid bone in these cases is not involved by any acute disease process. As a rule, the bone shows evidences of sclerosis.

The subject of primary jugular bulb thrombosis was brought to the attention of the medical profession by McKernon in 1904.[1] At that time he referred to the relationship which exists between an apparently unexplained fever and an acute otitis media. The diagnosis, difficult at

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From the Otological Service of Dr. I. Friesner and Laboratories of the Mount Sinai Hospital.

The illustrations appearing in this article were used through the courtesy of Dr. J. G. Druss.

1. McKernon, J. F.: Primary Jugular Bulb Thrombosis in Children, New York M. J. **82**:4, 1905.

times even now, was still greater at the time of McKernon's report, as his work antedated the important blood culture studies of Libman in 1906.[2] The technical aspect of the subject of blood culture as developed in the Mount Sinai Hospital by Libman and by Shwartzman [3] and the recent differential blood culture investigations of Ottenberg, to which more extended reference will be made, have contributed still further to the early recognition of this condition.

ANATOMIC AND PATHOLOGIC CONSIDERATIONS

A brief review of the anatomic relationships of the region should aid in a clearer understanding of the mechanism of bacterial invasion of the jugular bulb. The jugular bulb is the connecting link between the sigmoid sinus and the internal jugular vein. It is a dilated reservoir which lies under the floor of the middle ear. In order to reach the jugular vein, the bulb passes through the postero-external compartment of the jugular foramen. The upper extremity of the bulb is rounded and is called the dome of the jugular bulb. It lies in the jugular fossa, which is a rounded cavity hollowed out of the under-surface of the petrous pyramid. When the bulb is very small, it is separated from the floor of the middle ear by a comparatively thick layer of bone, sometimes 1 cm. thick. This may consist of compact bone; however, it more frequently contains a few cells. The cells in this layer of bone may become diseased and transmit infection to the bulb. When the bulb is very large, it reaches into the middle ear. In these cases the floor of the middle ear is thin and may even present dehiscences. The bulb, so situated, may be injured in the performance of a paracentesis of the drum membrane. Because of the lesser density of its bone covering in children, the bulb is more susceptible to infections arising in the middle ear during the early years of life.

The anatomic factors which predispose to involvement of the bulb are as follows: The bony partition separating the cavity of the middle ear from the jugular bulb is often extremely thin. An unusually high dome or roof of the bulb may encroach on the floor of the middle ear. Frequently there are dehiscences in the floor of the tympanic cavity, and as a result there may be direct contact between the floor of the middle ear and the dome of the bulb. The mastoid process in young children not infrequently shows an arrested development or a congenital absence of cells.

Under these conditions, an active purulent infection of the middle ear may invade the blood current after attacking the bulb, the pathway

2. Libman, E.: Tr. Am. Otol. Soc. **10**:315, 1906; Tr. Ninth Internat. Otol. Cong., 1912, p. 127.

3. Crohn, B. B., and Shwartzman, G.: Positive Blood Cultures in Non-specific Ulcerative Colitis, J. Lab. & Clin. Med. **14**:722 (May) 1929.

of infection being through the small communicating veins and lymphatics or by a destructive process through the bone. An infection in the tympanic cavity may first involve the carotid plexus of veins situated along the anterior tympanic wall, from which the infection may pass directly into the bulb and thus produce a primary bulb thrombosis. It is important to remember that the jugular bulb may become directly infected as a result of an acute inflammatory process in the middle ear without evidence at any time of a discharge from the middle ear.

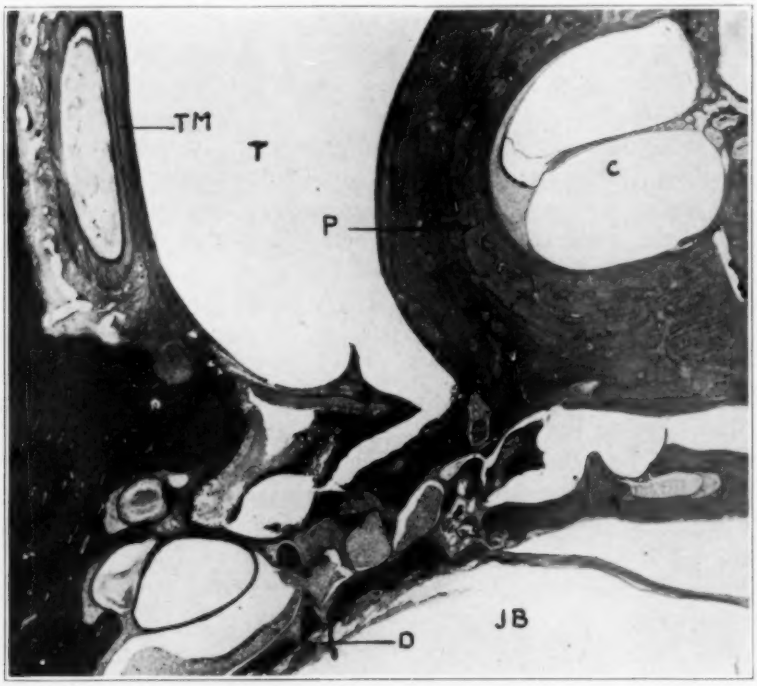

Fig. 1.—Dehiscences in the roof of the jugular bulb. *JB* indicates the jugular bulb; *D*, dehiscences; *TM*, the tympanic membrane; *P*, a promontory, and *C*, the cochlea.

In those cases in our series in which the extent of the disease was limited, especially in the early stages, that portion of the sigmoid sinus just above the bulb showed pathologic changes. Even when the entire sigmoid sinus was involved, the proximal end always evidenced a further advanced phlebitic process. In the cases in which occluding or mural thrombi were present, the sinus wall was usually found to be grayish and thickened. In those instances in which the lumen of the sinus was filled with pus, the sinus wall, aside from thickening, appeared yellowish or even reddish.

CLINICAL FEATURES AND COURSE

A review of the age incidence discloses that the illness is limited to children and young adults. The youngest patient was 6 and the oldest 21 years old. In the majority of the cases, the ages ranged between 7 and 14. In the twenty cases, there were eleven female and nine male patients.

The onset may be insidious, the aural history being so indefinite as to escape the attention of those under whose observation the patient first comes. In one instance, there was a history of fleeting pain in the

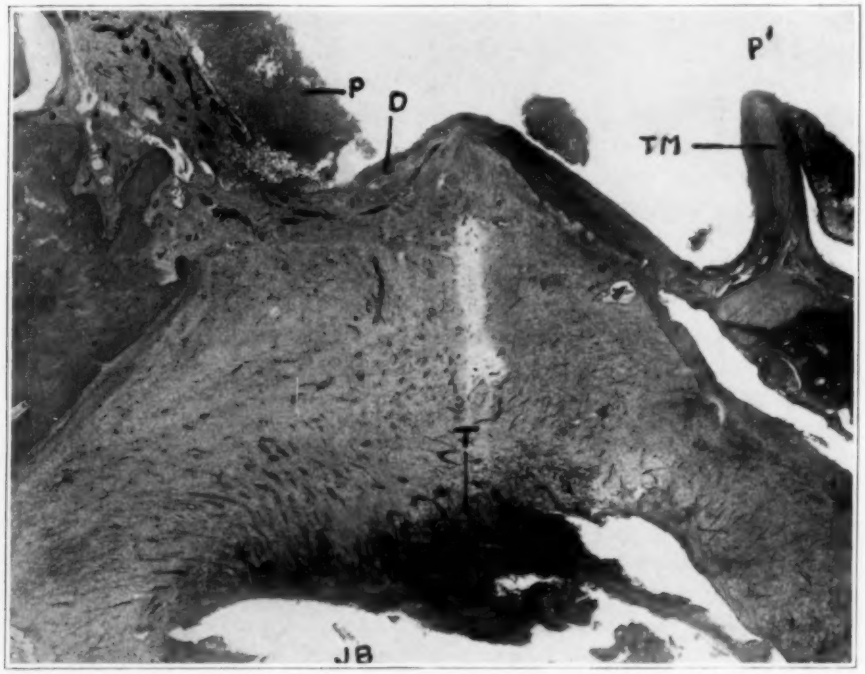

Fig. 2.—Organized thrombus in the jugular bulb. *JB* indicates the jugular bulb; *D*, a defect in the roof of the jugular bulb; *TM*, a remnant of the tympanic membrane; *P*, purulent exudate in the middle ear; *T*, a thrombus, and P^1, a perforation of the drum.

right ear of one day's duration two months previous to admission; at no time was there a discharge from the middle ear. A diagnosis of primary jugular bulb thrombosis was finally made, and the relationship between the prolonged general infection and a negligible otitic history was established and confirmed at operation. In another instance, the possibility of a sinus thrombosis was considered when hematuria, due to a focal infection of the kidney, appeared. The infection of the middle ear was mild. The diagnosis of primary jugular bulb thrombosis was made on the report of a positive blood culture.

There is always a history of an acute otitis media, although the preexisting inflammation of the middle ear may have been so slight as to cause no perforation of the drum membrane. The infection may have resolved in a few days. In the majority of instances, aural examination disclosed evidence of a subacute purulent otitis media or of a beginning resolution of the middle ear without involvement of the mastoid. The drums are flat and thickened, and the short process or hammer handle may be evident. The hearing, as a rule, is only moderately impaired.

A symptom of particular importance is a fluctuating temperature between 99 or 100 and 104 F. or higher. Frequently, the fever is low, the temperature being 100 or 101 F. for a few days, followed by a rapid rise and fall. Most of our patients had such a history of two or three weeks' duration. If rapid fluctuations in fever continue, pallor becomes quite evident, and the tongue appears white and dry.

The characteristic intermittent type of temperature is not always present in these cases. There may be neither declines to the normal nor high elevations. In the toxic cases there is often a continuously high fever like that of pneumonia. Many of our patients have had a temperature of this type at the onset, followed by a more or less characteristic septic temperature. In some cases the daily temperature does not rise above 102 or 103 F. It may descend to normal or subnormal or may drop only 1 or 2 degrees every day. The temperature is rarely normal all the time. This is especially likely to occur in cases in which the thrombus is sterile and has undergone partial or complete organization. Continuous fever lasting for a day or more at a moderate height or a normal temperature may be followed by a fresh rise on account of the development of metastases.

The majority of these patients do not have a distinct chill. They are more likely to experience chilly sensations.

Daily estimation of the hemoglobin percentage and red blood cell count usually reveals a progressive secondary anemia. The hemoglobin may drop to 35 or 40 per cent. The blood count, as a rule, shows a leukocytosis, from 12,000 to 20,000, and a polymorphonucleosis, from 75 to 85 per cent. A number of the patients had either a normal or a low white blood cell count. It is important to have in mind the fact that a leukopenia may exist during the active stage of a general infection with Streptococcus haemolyticus. Study of the young and staff cells during the course of an otitic infection furnishes important evidence of the intensity of the existing infection, and the cytoplasmic changes of the polymorphonuclear cells (toxic granules and vacuolization) are extremely important from the prognostic standpoint.[4]

4. Rosenwasser, H., and Rosenthal, N.: The Blood Picture in Otitic Infections, Arch. Otolaryng. **14**:291 (Sept.) 1931.

Changes in the ocular fundi were present in thirteen of our twenty cases, in seven before operation. The most marked changes in the fundus were observed after operation, this being so in eleven cases. We believe that these alterations were due to the ligation of the internal jugular vein and circulatory changes. Papilledema was observed to persist for a considerable period in several cases, but recession to normal finally occurred.

A striking negative finding in all these cases was the absence of petechiae or any other cutaneous manifestations.

The clinical picture of an otogenic general infection is varied. The good general condition of the patient is apt to be misleading. At the onset the patients usually do not look or feel sick. It should also be understood that the so-called atypical cases are more numerous than the typical ones. One must realize that each case is a problem in itself and that the picture is rarely complete.

The following signs associated with a definite aural history should arouse one's suspicion of the presence of a bulb thrombosis, even before the report of a positive blood culture: pallor, dry tongue, an enlarged spleen, enlarged and tender lymph glands at the angle of the jaw and early changes in the ocular fundi. The diagnosis can be established in the presence of a general infection unaccounted for by any other demonstrable lesion, by a history of acute otitis media and by a posifive blood culture.

BACTERIOLOGY

In this series of twenty cases of primary jugular bulb thrombosis observed during the past eleven years, in nineteen of which blood cultures were taken before operation, positive findings were obtained in all of the cases. Streptococcus haemolyticus (beta streptococcus, Brown) was isolated in each instance. In one case in which the blood culture was taken only after operation, Streptococcus nonhaemolyticus (gamma streptococcus, Brown) was reported. This higher percentage is at variance with the reports of many otologists who obtain a considerably lower number of positive results. Bacteremia is probably present at some time in every case of otogenic general infection, but it is not always possible to demonstrate it. When there is a bacteremia, the blood usually contains only one type of organism, even though several types of bacteria may be obtained from the middle ear, mastoid or even the thrombus. The experiences of Friesner [5] and of one of us (Maybaum) have confirmed the original observation of Libman,[2] that pneumococcic infections, excepting the type III organism, do not seem to give rise to sinus thrombosis.

5. Friesner, I.: Infections of the Middle Ear, Arch. Otolaryng. **14**:257 (Sept.) 1931.

Thirteen of our twenty cases continued to show a positive blood culture following operation, in seven on only one occasion and in six a number of times. The finding of a positive blood culture after operation is not necessarily an indication for further operation. Such a positive finding immediately after operation is not unusual and may be expected as a result of the trauma. When occurring at this time, the bacteremia may be due to incomplete operative intervention; or if a thorough operation was performed, other inaccessible channels (the superior and inferior petrosals) may be the pathway of invasion. In the presence of a persistent bacteremia, the general condition of the patient should be considered before resorting to operation again, provided the surgeon is reasonably certain that the focus was dealt with thoroughly.

DIFFERENTIAL BLOOD CULTURES

Valuable aid in the diagnosis of sinus thrombosis has resulted from the recent blood culture studies of Ottenberg.[6] The number of bacteria in the blood of the suspected internal jugular vein is compared with the number in the blood of the opposite internal jugular vein and, more recently, also of the vein of the arm. It was thought that the vein draining the lesion should show the largest number of bacteria. A surprising observation was made, however. In more than one half of the cases of sinus thrombosis in which bacteria were found in the blood, the preponderating number of bacteria was found not in the blood of the internal jugular vein on the same side as the thrombosed lateral sinus and directly draining it, but in the internal jugular vein draining the opposite or normal sinus. The most likely explanation for this phenomenon is that in these instances bacteria were growing chiefly at the upper end of the thrombus, and entered the circulation by way of the anastomoses to the opposite unobstructed jugular vein. The circulation at the proximal end of the jugular vein and the bulb on the diseased side is far less active than that at the torcular end. In every one of the cases in which the preponderating number of bacteria was found on the normal side, an obliterating thrombus was found on the diseased side at operation. In those cases of obliterating thromboses in which the preponderance of bacteria was found in the vein of the diseased side, it is probable that the bacteria were being liberated chiefly from the lower end of the thrombus.

In cases of sinus phlebitis without complete obstruction to the normal blood current in the sinus, one would expect to find the larger number of bacteria in the homolateral jugular vein. Ottenberg found that this was the case in four such instances.

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6. Ottenberg, R.: Differential Blood Cultures, J. A. M. A. **94**:1896 (June 14) 1930.

In a case in which the question of sinus thrombophlebitis has arisen, the finding of a much greater number of bacteria in the blood of one internal jugular vein than in that of the other, especially as compared to the peripheral circulation, confirms the diagnosis of infection of the sinus wall. However, it is impossible from the bacterial count alone to tell on which side there is infection, or whether there is a mural or an occlusive thrombus. The Ottenberg differential blood culture has been helpful in the diagnosis of obscure cases of general infection with evidence of otitis media.

COMPLICATIONS

Of our twenty cases, complications developed in fifteen, in six before operation and in nine after operation. Metastases usually occur after the thrombophlebitis has existed for some time. In the very severe and the mild infections, as a rule, metastases do not develop. In the moderately severe infections, metastases develop if the infectious process in the sinus is allowed to continue. Every metastatic focus does not go on to abscess formation. The formation of metastases or contiguous extension of the disease process is principally recognized by a continuance of general manifestations (intermittent fever, chills and the like) after the purulent foci of the ear and venous channels have been surgically treated. Local pains often point to the site of metastatic formation.

The joints and subcutaneous tissues are more commonly affected than the viscera. Of the latter, the meninges, brain and lungs were more frequently involved, but any other organ may be the seat of metastases.

Involvement of the joints usually takes the form of a periarticular or an epiphyseal inflammation without suppuration. Metastases in the soft parts are readily recognized. They usually manifest themselves as painful and reddened swellings which sometimes break down, but frequently resolve without suppuration.

Extension of the suppurative process to the meninges and brain gives signs of meningitis or an abscess of the brain. Metastases should be looked for carefully at an early stage, not forgetting the infrequent sites (the kidneys, spleen, liver and other organs).

Pulmonary metastases are usually indicated by the clinical picture of an infarct. They may exist without giving symptoms, as they may cause trouble only after pleural involvement in the region of the abscess. As a rule, there is a complaint of sharp pain in the chest, cough and sometimes bloody expectoration. The condition may subside or advance with the formation of an abscess of the lung. In other instances of pulmonary metastases, there may be numerous embolic foci scattered throughout both lungs, often evidenced by the presence of râles, which are evanescent and of a changeable character.

DIFFERENTIAL DIAGNOSIS

It is, of course, essential that other disease conditions presenting symptoms similar or even identical with those induced by thrombophlebitis should be definitely ruled out. This is a difficult task in an infection such as primary jugular bulb thrombosis, which frequently has so insidious an onset and course.

For the purpose of differential diagnosis, the following infections are among the more common conditions which should receive consideration: acute tonsillitis, pneumonia, erysipelas, acute nasopharyngeal infections and pyelonephritis. Any of these conditions, when associated with an acute suppuration of the middle ear, for a time may give rise to considerable confusion as to the exact diagnosis.

Acute follicular tonsillitis may be mistaken for jugular bulb thrombosis when the tonsillitis complicates suppuration of the middle ear. High temperature and bacteremia may occur with tonsillitis. Throat symptoms, such as local and radiating pain and dysphagia, sometimes occur with sinus thrombosis, especially with bulb thrombosis if there is involvement of the glossopharyngeal nerve. It may be necessary to wait until the tonsillitis subsides before making the diagnosis.

Pneumonia may have to be differentiated from jugular bulb thrombosis. It may be present with few signs in the chest. When there are pulmonary metastases in sinus thrombosis, the physical signs in the chest are confusing. If the organisms found in the blood in pneumonia are pneumococci of types other than type III, the differential diagnosis is easy as only the type III pneumococcus is found in the blood stream in cases of sinus thrombosis. Roentgen examination of the chest gives valuable information. Pyemic symptoms in pneumonia are present only if the infection is of a severe type and has been extensive from the start. One should not be content with uncertain pulmonary findings or with the opinion of the presence of a central pneumonia which cannot be verified by auscultation or roentgen examination. Due consideration must be given to the ear as a causative factor in the presence of pyemic manifestations in such cases.

Erysipelas, developing in the course of otitis media or external otitis, may for a time present difficulties in diagnosis. The temperature is usually high, and the rise may be continuous, remittent or intermittent. If redness of the skin appears within a day or two after the onset of the fever, the diagnosis is easy, but sometimes the redness does not appear for five days or more. Rarely a blood culture may be positive in the early stages of the disease, but it soon becomes negative.

Cervical adenitis occurring with suppuration of the middle ear often confounds the clinical picture, especially in children. This condition frequently causes the temperature curve to simulate the picture produced

by otogenic general infection. There may be tenderness along the jugular vein. Although the temperature may be quite high over a considerable period in children who have a cervical adenitis, the patients usually do not look or act as sick as those with otogenic general sepsis. Cervical adenitis usually does not cause a bacteremia. Rarely cervical adenitis may cause thrombosis of the jugular vein with extension to the sigmoid sinus.

Acute nasopharyngeal infections and septic sore throat may give rise to high temperatures which may be confused, in the presence of an infection of the middle ear, with jugular bulb thrombosis. These infections are usually accompanied by cervical adenitis. The blood culture may be positive.

Pyelonephritis is occasionally complicated by a suppuration of the middle ear. The condition can readily be diagnosed by examination of the urine. Despite rises in temperature over a long period, the general condition of these patients is good.

It must be evident from this brief discussion of the differential diagnosis of jugular bulb thrombosis that the problems are so varied and difficult at times that otologists should have the constant cooperation of a pediatrician or an internist from the very onset of the disease. Also, blood culture and bacteriologic studies offer invaluable assistance in the solution of these problems.

PROGNOSIS

The mortality in our group of cases of primary bulb thrombosis is strikingly low; only two of the twenty patients died. We found that children tolerate operative intervention rather well. Due regard, however, has to be given to early operative intervention, the duration of the operative period, thoroughness in operative technic and the use of intravenous injections of saline solution during the operation to prevent shock.

TREATMENT AND MANAGEMENT

The indications for treatment of otogenic general infection may be grouped under five heads:

1. Performance of a simple mastoidectomy in order to obtain access to the sinus and jugular bulb.

2. Evacuation of the thrombus and removal of as much of the infected sinus wall as is necessary to prevent the spread of the infection in the sinus. The same refers to the internal jugular vein when the disease has spread to this vessel.

3. Blocking of the main venous channels through which the general infection is being carried into the general circulation, that is, ligation

or resection of the internal jugular vein and obliteration of the sinus wall beyond the diseased area.

4. Increase of body resistance by improving the quality of the circulating blood, that is, the employment of blood transfusion.

5. Treatment of metastases.

To be effective, surgical methods must be instituted early. Briefly, our procedure is as follows: The diagnosis having been made, the mastoid is rapidly exenterated, the sinus is exposed and followed back until it appears to be normal, and a low ligation of the internal jugular vein is made through a horizontal incision. The sinus is then incised after plugs have been properly placed at the torcular and bulb ends. If the condition of the patient permits, the sigmoid groove is followed to the bulb; the latter is then curetted and drained. It is of utmost importance to obtain free bleeding from the torcular end of the exposed sinus.

In a majority of instances operative procedures on the bulb are unnecessary. If sepsis continues despite thorough surgical measures directed to the torcular end of the sinus and evidence points to suppuration in the jugular bulb, the indications are to deal with the focus in the bulb. The question of the presence of suppuration in the bulb is determined by one's ability to cause pus to well up into the mastoid cavity from the bulb on pressure over the tender swelling which is present at the angle of the jaw.

The formidable surgical procedures advised and described by Voss, Grunert and Tandler have not been found necessary in our experience. Besides being extensive, they entail a certain amount of danger to the facial nerve. Ordinarily we expose the sigmoid sinus to the posterior bony canal wall and then curet the remainder of the sigmoid sinus and as much of the bulb as we can reach. Nothing more was done in all but two of the cases. In these two cases, the procedure of doing a complete operation on the sigmoid sinus answered the purpose. This method of exploring the bulb is based on the early steps in the operations of Voss and Grunert. Because of its comparative simplicity and the rapidity with which it can be performed, together with the entire freedom from danger of injury to the facial nerve, this modification recommends itself as an ideal method for exploring the bulb. It differs from those previously referred to in that it is less radical and yet is thoroughly efficacious in draining the bulb. When the sigmoid sinus approaches the posterior canal wall at a more acute angle than usual, the procedure may not be feasible. If pus can be pressed into the mastoid wound from the bulb, we find daily irrigations with surgical solution of chlorinated soda, carefully performed, efficacious. In the majority of instances this procedure is all that is necessary to effect a favorable result.

Blood transfusions should not be used indiscriminately as a therapeutic agent to combat a general infection of otitic origin. Patients in a serious condition as a result of general infection of comparatively long duration withstand operations better if given a transfusion before operation. In the majority of instances a blood transfusion is given immediately after operation. As to subsequent transfusions, we have had recourse to these only if the general condition, especially as indicated by a decreasing hemoglobin and red blood cell count, warrants it.

Summary of Cases of Primary Jugular Bulb Thrombosis

General History:		
Age incidence		6 to 21 years
Age average		10 years
Females		11
Males		9
Aural History:		
Bilateral suppuration of middle ear		5
Acute purulent otitis media		19
Acute otitis media (no otorrhea)		1
Types:		
Acute		7
Resolving		12
Acute otitis media (no otorrhea)		1
Repeated attacks of otitis media		8
General Symptoms:		
Chills		8
Chilly sensations		6
Septic temperature (typical or with variations)		20
Headache		8
Vomiting		3
Enlarged spleen		13
Cervical glands (enlarged and tender)		11
Skin—petechiae		0
Fundi:		
(1) Normal throughout		7
(2) Normal before operation; changes after operation		6
Papillitis	1	
Peripapillitis	2	
Optic neuritis	2	
Margins indistinct	1	
(3) Changes before operation; no postoperative advance		2
Slight elevation	1	
Papilledema	1	
(4) Changes before operation; further postoperative advance		5
Early optic neuritis increased to papilledema	3	
Haziness of margins to optic neuritis with peripapillary hemorrhages	1	
Papilledema increased with postneuritic atrophy	1	
Operative Findings:		
Sclerosed mastoid		7
Cellular mastoid		13
Mastoid not involved		20
Sinus plate intact		20
Sinus wall diseased		20
Sinus contents		
Obturating thrombus		9
Fluid pus		5
Thrombus not demonstrable		6
Bulb		
Bleeding absent		13
Bleeding slight		1
Bleeding free		6
Torcular end		
Bleeding absent		2
Bleeding slight		4
Bleeding free		14

Summary of Cases of Primary Jugular Bulb Thrombosis—Continued

Complications:				
Preoperative cases 6	Viscera 3		Acute focal glomerular nephritis	1
			Subcortical abscesses of kidney	1
			Lungs—embolic foci	1
	Joints 2	Periarthritis	Hip	
			Ankle	
	Subcutaneous 2	Abscess	Buttocks	
			Thumb	
Postoperative cases 9	Viscera 4		Pulmonary infarct	1
			Meningitis (suppurative)	2
			Temporosphenoidal abscess	1
			Gradenigo syndrome	1
	Joints 3	Periarthritis	Ankle	2
			Shoulder	1
			Metacarpal	1
		Spondylitis	Dorsal	1
			Cervical	1
		Thoracic spine—abscess		1
	Subcutaneous 3	Abscess	Forearm	
			Thigh	
			Buttocks	
Blood Cultures:				
Positive before operation, Streptococcus haemolyticus (total number taken)				19
Positive only after operation, Streptococcus anhaemolyticus (taken only after operation)				1
Positive after operation				13
Positive only once			7	
Positive repeatedly			6	
Result:				
Patients recovered				18
Patients dead				2

REPORT OF A CASE

Since our cases were studied, a patient with primary bulb thrombosis was admitted to the pediatric service in a moribund condition. The demonstration of a thrombus confined to the jugular bulb without involvement of the sinus on postmortem examination is of sufficient importance and interest to warrant a report of the case.

History.—P. C., a girl, aged 8 years, was ill with an attack of the grip with high fever four weeks before admission to the hospital. Pain was present in the left ear at the onset, and a myringotomy was performed two days later. No discharge emanated from the ear, but the pain continued for four days more. Shortly after the disappearance of the pain, the patient had a chill which lasted one-half hour, and since then she had had five similar chills, the last one occurring three days before admission. For the last three weeks prior to admission, the patient had had an intermittent fever, the temperature rising in the afternoon and falling to normal at night.

About ten days before admission, the patient experienced pain, heat and tenderness in the right knee, which lasted for one day and then decreased. The following day the left elbow was similarly affected, but this process progressed. A week later the left knee was involved, and four days before admission the lower dorsal spine was painful on extension and motion.

Positive Physical Findings.—The patient was acutely ill and lethargic; the respirations were rapid and deep. The right ear was normal. The drum of the left

ear was red and thickened; there were no landmarks and no discharge. There were no changes in the canal wall and no tenderness of the mastoid. The liver was palpable for four fingerbreadths. The spleen was enlarged; there was tenderness in both lower quadrants. There were swelling and tenderness of both hip joints. There was a restriction of motion of both knees and the left elbow.

Laboratory Data.—A blood count showed: hemoglobin, 65 per cent; red cells, 3,850,000, and white cells, 26,450, with polymorphonuclears, 93 per cent (5 per cent staff cells), and lymphocytes, 7 per cent; there was a marked toxic granular degeneration. Blood culture was reported as yielding Streptococcus haemolyticus in all mediums, 150 colonies to the cubic centimeter.

Roentgenograms of the chest and joints were negative; those of the mastoids were unsatisfactory, and those of the nasal sinuses showed a slight cloudiness of the right antrum and ethmoids.

Fig. 3.—Specimen showing a thrombus in the jugular bulb. *SS* indicates the normal sigmoid sinus; *T*, a thrombus in the jugular bulb, and *IAM*, the internal auditory meatus.

Opinion.—The diagnosis was general infection of otitic origin—primary jugular bulb thrombosis. The child was in a too critical condition for surgical intervention.

COMMENT

This case is of unusual interest in that on postmortem examination a partly organized purulent thrombus was found confined to the jugular bulb without involvement of the sigmoid sinus (fig. 3). The necessity for exposing the sigmoid sinus to the bulb in cases of suspected jugular bulb thrombosis is apparent, even though the sigmoid sinus appears to be normal. The case clearly demonstrates the various points brought out in the paper as to the etiologic factors, clinical course and pathology of primary jugular bulb thrombosis.

CONCLUSIONS

1. A study of twenty cases of primary jugular bulb thrombosis observed over a period of eleven years is presented.

2. Certain anatomic factors, such as a thin bony partition separating the middle ear from the bulb, a high dome and dehiscences, predispose to involvement of the bulb.

3. In children or young adults with a history of an acute infection of the middle ear, a fluctuating fever, a progressive anemia, changes in the ocular fundi and evidences of a general infection otherwise unaccounted for, primary jugular bulb thrombosis should be suspected.

4. The additional presence of a positive blood culture establishes the diagnosis.

5. A majority of the cases follow a characteristic course, suggesting a clinical entity.

6. Blood culture studies are of paramount aid in diagnosis. Those taken before operation (nineteen of the twenty cases) were all reported positive for Streptococcus haemolyticus.

7. The following conditions must be considered in the differential diagnosis: acute tonsillitis, pneumonia, erysipelas, acute nasopharyngeal infections and pyelonephritis.

8. In the surgical treatment, occlusion of the main channels of systemic invasion, especially of the torcular end of the diseased sinus, is of prime importance.

9. An operation on the jugular bulb should be performed only if there is a continuance of bacteremia and symptoms, general or local, after the main channels have been obliterated. Extensive operations on the jugular bulb are seldom indicated.

10. For a favorable outcome, early diagnosis and prompt surgical intervention, properly directed, are imperative.

1070 Park Avenue.

1235 Park Avenue.

Clinical Notes

CURE IN A CASE OF CEREBROSPINAL RHINORRHEA

NOAH FOX, M.D., CHICAGO

The case in question is of twofold interest, first because of its occurrence during the course of a cold, and second because of the simplicity of treatment which resulted in an apparent cure.

REPORT OF CASE

Miss B. L., aged 33, gave the following history: In the early part of March, 1930, she contracted what appeared to be an ordinary cold in the head, of possibly a little more severity than she had ever previously had. Especially prominent were the symptoms of sneezing and nasal discharge. No headache had ever been present. The cold lasted for about two weeks, but she noted that after all other symptoms had subsided, a clear fluid continued to drip from her left nostril, particularly when she bent her head forward. The nasal drip had been present for about four weeks when the patient was first examined. Nasal examination gave entirely negative results, all of the changes from the previous cold having disappeared. However, a clear watery fluid was found to be dripping from the nose, so that a 20 cc. test tube was easily filled in about thirty minutes. This fluid was clear, having a specific gravity of 1.008. When acetic acid was added, no precipitate appeared. Boiling failed to produce a coagulum, but the addition of magnesium sulphate to the point of saturation caused precipitation. The addition of nitric acid caused a precipitate to form, which disappeared when the solution was boiled. With a trace of copper sulphate and excess of potassium hydroxide, the solution turned pink. When boiled with Fehling's solution, a reduction occurred.

In order further to ascertain the character of the fluid, 5 cc. of 0.6 per cent indigo carmine was injected into the spinal canal. A bluish-green coloration of the nasal drip appeared after fifteen minutes and continued for about five hours. Nasal examination made at this time revealed that the colored material was dripping over the lateral wall of the left nasal cavity in the region of the carina and the agger areas. It was assumed that the separation in continuity of the cribriform plate had occurred anteriorly, where it might be easily accessible to intranasal treatment. In seeking an etiologic factor, several stereoscopic plates, both in the lateral and in the anteroposterior positions, were taken, but appeared negative. The eyegrounds and visual fields appeared normal. The obesity of the patient suggested hypophyseal disease, but no evidence of this had occurred. An attempt was made to set up a fibrosis in the area involved, which might result in occlusion of the separation, so the roof of the nose was swabbed every two weeks with a 20 per cent solution of silver nitrate. After six applications fibrous bands began to appear, and the discharge diminished until it entirely stopped, four months after treatment was begun. It has remained dry for eighteen months. The patient is apparently cured, without either nasal or intracranial symptoms.

From the Department of Otolaryngology, University of Illinois.

COMMENT

The injection of a dye into the spinal fluid gives one a clue as to the location of the fracture and the practicability of this type of treatment. In carrying out the treatment, however, one must be quite sure of the absence of intracranial pathologic changes that might be increasing the cerebrospinal pressure. The danger of stirring up a meningitis should also be kept in mind.

ASPIRATION OF FLUID FROM THE LUNG

An Improved Method

ALFRED LEWY, M.D., CHICAGO

The use of the catheter intratracheally for aspiration of fluid has long been in use, especially by obstetricians for resuscitation of the new-born. In order to make the method less haphazard and to control to a considerable extent the passage of the catheter into either bronchus as desired, I have used the following method:

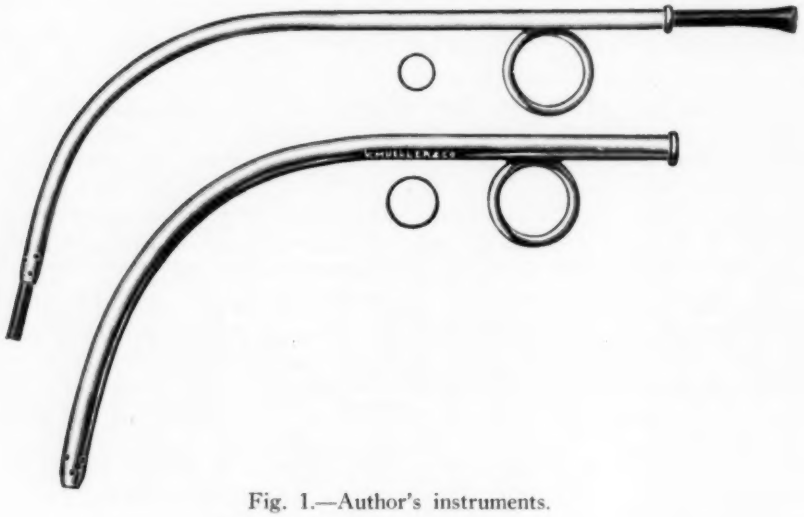

Fig. 1.—Author's instruments.

After cocainization, a Mosher metal "life saver," with the tip cut off to allow the passage of a waxed linen catheter, is passed through the glottis into the trachea by sense of touch, as in ordinary intubation of the larynx; a waxed linen ureteral catheter of the largest possible size is then passed through this. Tilting the head and neck and the body slightly to one side or the other permits the catheter to engage in the opposite bronchus, and it is then slipped down as far as it will go, or as far as is desired. A suction bulb, syringe or regular apparatus for suction can be attached. For infants and small children the ordinary waxed linen urethral catheter is long enough.

This method of aspiration is applicable in cases of pulmonary abscess, bronchiectasis and postoperative obstructions, even possibly in cases of pulmonary edema. It may in many cases take the place of bronchoscopy, in the absence of a skilled bronchoscopist. An instrument of smaller size has also been devised for use in infants, and I hope to try it in cases of bronchial hypersecretion. Approximately the same method may have been used by others. Its usefulness should be extended.

The roentgenogram was taken in a case of abscess of the left lung and distortion of the trachea, with the catheter in place in the left bronchus. Considerable pus was evacuated; the patient had previously been treated by bronchoscopy.

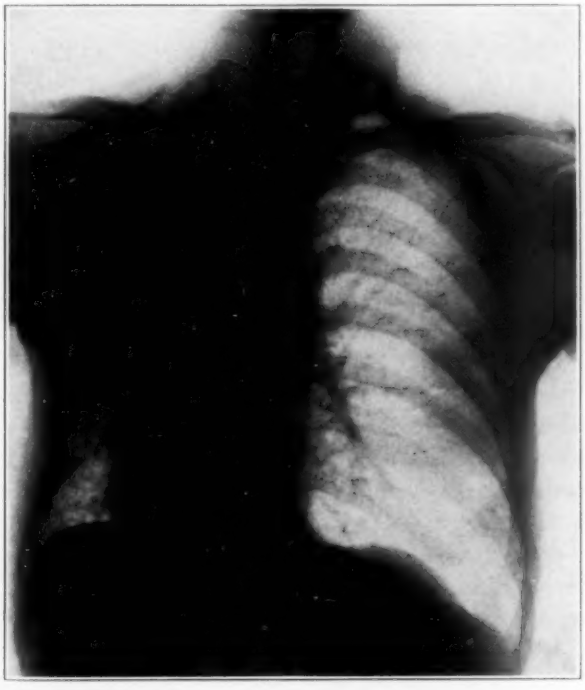

Fig. 2.—Catheter in place in a case of abscess of the left lung.

These instruments were made for me by V. Mueller & Company, Chicago.

NOTE.—Since this article was prepared, it has come to my attention that Dr. M. J. Mandelbaum, of New York, has developed a method and instrumentarium for the same purpose, but he has not published a description of his method and the details are not known to me.

25 East Washington Street.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology.

FUNCTIONAL TESTS OF HEARING

ROBERT SONNENSCHEIN, M.D.
AND
NORMAN LESHIN, M.D.
CHICAGO

During the year Oct. 1, 1931 to September, 1932, the number of papers on functional testing of hearing and allied subjects has not been large. In the German and French literature there was a paucity of articles on this subject; in fact, we found none in French and only a few in German. The preponderance of English and especially American contributions has now been noted for several years. It is natural that on a subject like functional testing of hearing in which the fundamental tests were devised many years ago, it is difficult indeed to bring out much that is new. It is, however, an indication of the earnestness of the workers in the field of otolaryngology that they endeavor not only to apply the standard tests, but to delve into the problems of air and bone conduction, concerning the physics of which there is still considerable discussion.

Fowler [1] presented the pioneer efforts of the New York League fo. the Hard of Hearing in the scientific approach to the problem of the prevention of deafness. Every member is requested to answer a questionnaire on deafness in his family, and to submit a brief personal history of his ear trouble, with an audiometric examination of his hearing. Since little attention to deafness is given in the ordinary otologic clinic because of too many patients or lack of energy, a clinic was established at the Manhattan Eye, Ear and Throat Hospital for the children found affected in the League's survey of hearing in the schools. The object of this was primarily to stimulate otologists and the leagues in other cities to try a similar project to study the diseases of the ear causing deafness. At present there are six such clinics.

The scientific work is divided into clinical and laboratory. The clinical phase is carried out by taking a careful personal and family

1. Fowler, E. P.: Scientific Aspects of the Work of the American Federation of Organizations for the Hard of Hearing, Ann. Otol., Rhin. & Laryng. **40**:1036 (Dec.) 1931.

history. Otoscopy is made by the naked eye and the magnifying pneumatic speculum, and a sketch of the drum is made on a chart. Vertigo and tinnitus are investigated, and vestibular tests are made when indicated. The fistula test is done on every suppurating ear. Examination of the nose and nasopharynx is made and the patency of the eustachian tubes determined. If an obstruction of the tube is suspected, the inflation is repeated with the head bent sharply toward one side and then the other. This determines the efficacy of the tubal abductors, since stretching the muscles of the neck permits the abductor tubal muscles to open the lower end of the tube better.

The audiometric determination is made for air conduction at all frequencies and bone conduction up to 4,000 double vibrations. Fowler has discarded the lower tone limit and Rinne tests as they are unnecessary and misleading, since he feels that the actual tones in sensation units by air and bone conduction give more accurate and understandable measurements which are comparable at all frequencies. A better method for evaluating the hearing capacity for the Galton whistle is to blow it first before the open ear and then before the ear held closed. The normal ear will easily hear the sound even though it is closed, but many deafened ears will not and may then be classed as being deficient for higher tones. Calibrated tuning forks are used only by the alternate placement method, the only way air conduction and bone conduction may both be measured by the same instrument in comparable periods of decrement of the fork. The approximate loss of sensation units for the spoken voice is determined for the faint and medium whisper and the faint, medium, loud and shouted voice. If these are not heard at 15 inches, it means a loss of at least 30, 45, 60, 75, 90 and 100 sensation units, respectively. Fowler suggested the use of an air douche with nozzle well within the external auditory canal in place of the ordinary noise apparatus. This is much better since the noise is created near the drum membrane and not outside the head; thus the communication of so much vibration through and around the head to the ear being tested is avoided. Careful audiometric tests are made before and after every mastoidectomy. Stereoscopic roentgenograms of the mastoids and nasal sinuses made with repeated audiometric tests have shown the important factor the nose plays in the etiology of ear disease. A great majority of the children showed some involvement of the nasal sinuses, and these changes quite regularly accompany recurrent suppurative and nonsuppurative otitis media. The treatment of the nose is often more important than that of the ear. This should be remembered as one may neglect an opportunity for the cure as well as the possible prevention of deafness. A Wassermann reaction of the blood is taken in every case, and blood counts and chemical analyses are repeated at intervals. Fowler again repeated a statement

made previously that there is no greater variation in the calcium and phosphorus content of the blood in otosclerosis than in many other classes of ear disease. Where the Wassermann reaction is positive, the entire family is urged to submit to blood and otologic examinations. Routine tuberculin reactions are also noted.

A definite diagnosis is attempted in every case, a scheme being used that is printed on the history chart and then cross referenced. Each disease is tabulated in four age groups, 1-9, 10-15, 16-21 and 21-all inclusive.

The pathologic research is done mainly at the College of Physicians and Surgeons. Animal experiments, especially with vitamin deficiency diets, are being made and the temporal bones and autopsy material studied.

Fowler expressed the belief that every league for the hard of hearing should cooperate with otologists to establish and carry on a clinic for the treatment and study of deafness and diseases of the ear.

The problem of the child who is hard of hearing [2] has become more important and its effects more far-reaching since the advent of the audiometer, special surveys by competent otologists, cooperation of various associations and the granting of funds by foundations to carry on this work. Over 10 per cent of our future citizens show a physical defect in hearing. With the use of the 4 *A* audiometer, large groups of children can be efficiently tested, the results promptly tabulated and hearing defects aggregated. Forty children can be tested simultaneously in twenty minutes. After the initial test children with borderline cases are retested with the 2 *A* audiometer and an audiogram made. A careful otologic examination and a diagnosis are also made. Notes to the teacher and parents are sent for the proper disposal of the case and a further check-up later, to see whether the proposed suggestions are carried out. This procedure of testing the whole school should be done yearly.

The outstanding conclusion of the report of the committee for the testing of the public school children of the United States was the urgent need for standardized methods for the detection of loss of hearing, disposition and the follow-up of the children having hearing defects. The enlistment of groups of experienced otologists to conduct special surveys was then undertaken, and the cooperation of national associations of otologists, teachers of the deaf and organizations for the hard of hearing was requested.

The pedagogic classification of the child who is hard of hearing and his educational development is more important than his clinical status.

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2. Goldstein, M. A.: The Hard of Hearing Child, Laryngoscope **41**:733 (Nov.) 1931.

This pedagogic classification is dependent on the age, degree of defective hearing and acquired fluency of speech.

There are several types of acquired total deafness: (1) that which develops before the development of speech, namely, before the age of 3, and (2) that which develops after the development of speech. The children in the first group should be trained as the child who has never heard speech. Those in the second group should be given every opportunity to conserve speech and must be constantly drilled to maintain it and prevent the characteristic monotone of the congenitally deaf child. The further classification is made according to the distance conversational voice is heard. Those who have deafness beyond 3 feet for conversational voice are given intensive and individual instruction in lip reading as early as 3 years of age. After the age of 8 or 9, when the speech is fluent and lip reading perfect, the child can be transferred to a normal schoolroom. For those children who are deaf beyond 12 feet for conversational voice, instruction in lip reading and voice conversation should be given intensively, but preferably in a graded school for children who are hard of hearing. Children with deafness beyond 20 feet for conversational voice should remain in the normal school room but be seated in favorable locations. Daily instruction in lip reading should also be given. Children with slight impairment of both ears, usually caused by otitis media and other conditions, should receive remedial or operative treatment, if advised by the otologists, but should remain in the normal schoolroom.

The greatest aid which may be found for the person who is hard of hearing is the efficient teaching and learning of lip reading. In addition, the lip reading teacher should also teach proper conversation and characteristics of the conversational voice.

Sommer [3] reported a series of twenty cases of primary glaucoma in which seventeen of the patients had impaired hearing of different degrees; the majority showed lesions of the inner ear. The ear involved was on the same side as the affected eye. The hearing test revealed fractionated air and bone conduction for the middle tuning forks, more frequently the latter in most of the cases. By fractionated conduction, he means that when the fork is held over the mastoid and the patient states he no longer hears it, he will again hear the fork when it is replaced without restriking it. This may occur three or four times. The same explanation holds for air conduction. This phenomenon is explained as due to a neurosis producing fatigue of the cochlear nerve. This fatigue symptom may also be present in early stages of eighth nerve involvement and may be the only finding of an incipient

3. Sommer, Ignaz: Ear Finding in Primary Glaucoma, Eye, Ear, Nose & Throat Monthly **2**:63 (March) 1932.

organic lesion of the cochlear nerve. Sommer stated that his findings are not definite proof of a relationship between deafness and primary glaucoma. Histologic study is necessary before a positive conclusion can be established. The fractionated air and bone conduction found are indicative of a cochlear lesion.

Taylor [4] repeated briefly the incidence of deafness in school children given by Fletcher and Fowler who report 14.4 per cent of the group tested as having deficient hearing. On this basis, with 24,000,000 school children in the United States, there are 3,000,000 with some hearing defect, and 340,000 of these have sufficient impairment of hearing to require instruction in lip reading. Many of the 3,000,000 children who are hard of hearing are considered stupid rather than deafened, and with proper aid are able to progress as well as the normal child. The indications for instruction in lip reading are based on the percentage loss found by audiometric examination. Those with a loss of from 9 to 12 per cent required no lip reading. Those with a loss of from 12 to 15 per cent required regular class work in lip reading several times a week in addition to the ordinary classroom work. A child with from 15 to 20 per cent loss of hearing requires special training. These children should be taught in smaller groups and may continue their association in play and some school work with the normal child. Those with 25 per cent or more loss of hearing belong in segregated groups with especially trained teachers in lip reading and speech correction.

Deafness in the school child is definitely related to the type of medical care received in the first ten years of life. In communities where economic conditions are fair, 15 per cent deafness is found, and in poorer environments the incidence is 25 per cent. In communities where medical care and expense is no economic factor, only 2 per cent of deafness is found. Seventy-five per cent of the cases of adult deafness are a direct result of inadequate medical care during the first ten years of life.

At present, special classes in lip reading are operating in forty-four city school systems, and sixty cities maintain evening classes in lip reading. Early discovery of hearing impairments is necessary for maximum benefits to be gained by proper instruction and medical care. In a great majority of cases, the child who is hard of hearing can receive his education in the regular public school provided certain special instruction can be given by his own teachers, and where necessary special classes in lip reading and speech are maintained for those who need them.

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4. Taylor, R. M.: The Hard of Hearing School Child, Laryngoscope **41**:753 (Nov.) 1931.

Coates [5] stated that the ideal way of preventing deafness is to control the initial factors which may later cause impairment of hearing to such a degree that deafness supervenes. Prevention of deafness also embodies detection of impaired hearing in the early stages when normal function can still be restored or further progress prevented. Therefore, the study of preventive measures falls into two classes, that of diagnostic and of therapeutic measures for detecting and curing the condition. Various agencies are necessary to accomplish this. These include research, education of the medical profession, health authorities, industrial managers, school authorities and the laity, propaganda to enforce education, proper legislation and arousing the mass of people to the realization of the great economic waste resulting from increase in impaired hearing.

Coates takes up in more detail the possible means of eliminating causative factors such as syphilis and the acute exanthemas. He also takes up the question of tonsillectomy and adenoidectomy, the problem of industrial noises and the avoidance of certain drugs such as the arsenical, salicylates and quinine, also alcohol and tobacco. The proper and skilful treatment of the nose and throat during the acute exanthemas may prevent infections of the middle ear. Also, skilful attention to an infection of the middle ear when it occurs may prevent or minimize the damage to the hearing. Careful and thorough exenteration of the mastoid during operation is also a factor in preventing irretrievable damage to the middle ear stimulus and hearing. Cleanliness of public bathing pools, proper instruction in blowing the nose and use of ear plugs where necessary are all factors in preventing infections of the middle ear.

For the detection of incipient losses of hearing, the aural examinations in school, especially more recently with the use of the 4 *A* audiometer where large groups can be tested at one time, have been of great value. Children with impairment of hearing should be sent for further study and treatment, if necessary. In addition, chronic suppurations should also receive proper attention and treatment, and if operative measures are necessary the type of operation should carefully be decided on, depending on the pathologic process. For this work a separate clinic, such as a "prevention of deafness" clinic, should be established to work in close cooperation with a properly conducted treatment and operative service. Close cooperation of the social service department is essential. To carry out a program of this nature, money, energy and many conscientious workers are necessary. Much help is being obtained

5. Coates, G. M.: The Prevention of Deafness, Ann. Otol., Rhin. & Laryng. **40**:651 (Sept.) 1931.

through the deafened themselves through their organizations and local leagues. These organizations should be given the active help and support of every otologist.

Bunch [6] stated that one of the criticisms of audiometer methods is the impossibility of eliminating the perception of the ear not being tested, because of the transmission by direct contact between the telephone receiver and the head. Various methods have been devised to eliminate this objection. The most recent is that of masking suggested by Bárány, who devised his "Laern" apparatus for this purpose. The scientific objection to this method is that no two apparatus produce sounds of the same pitch or loudness, and so they cannot be standardized. Since the masking principle is the most simple for this study, other appliances were sought. Bunch used the Western Electric 3 *A* buzzer audiometer which has an intensity-regulating dial for this purpose. A series of normal observers were first tested with and without masking the opposite ear, in order to substantiate the claim that a masking noise will to a large extent eliminate the hearing in the opposite ear. A soft rubber cushion was used to confine the sound energy more closely to the ear being masked. He found that a masking noise of 80 units loudness from the 3 *A* audiometer will cause the threshold of the opposite ear to be lowered from 10 to 15 units in persons with normal hearing. In a case of complete unilateral deafness, the masking of the deaf ear with the noise from the 3 *A* audiometer buzzer with 85 units of loudness had no measurable effect on the acuity of the hearing ear. In several cases of marked unilateral deafness, masking the hearing ear with from 65 to 85 units of loudness did not eliminate the residuum of hearing in the deaf ear.

The type of audiometric curve which has caused considerable discussion is that obtained in cases of certain unilateral deafness in which the totally deaf ear gives a curve which lies almost parallel to, and is but from 30 to 60 units below, that of the hearing ear. Such a curve for the deafened ear has been called a "shadow curve," because the hearing indicated by the curve is in reality a shadow of the hearing in the opposite or better ear. When such a curve is obtained, one should by some method of masking attempt to eliminate the possibility of the acuity of the better hearing ear entering into consideration when the thresholds for the poorer ear are being determined. This was done by Bunch in several of these instances, the 3 *A* buzzer and audiometer with loudness varying from 65 to 85 units as the case required being used, and no tones were found audible in the deaf ear at the maximum intensity produced by the audiometer, nor could the patient tell by the

6. Bunch, C. C.: Auditory Tests in Cases of Marked or Complete Unilateral Deafness, Ann. Otol., Rhin. & Laryng. **40**:748 (Sept.) 1931.

sensation of touch when the oscillating current was turned off or on in the receiver at the deaf ear. This method of masking will also bring out islands of residual hearing in some cases in which the usual shadow curve is obtained, denoting the possibility of total deafness.

Bunch expressed the belief that this method is the best for determining the presence, location and extent of the elements of residual hearing. He also stated and showed that by the proper selection of masking apparatus and with proper intensity control, it is possible to prevent the hearing of the good ear from entering into consideration when audiometric tests are being made in cases of marked unilateral deafness.

Drury [7] reported an audiometric study of two hundred and ninety-one persons with brain lesions from Dr. Harvey Cushing's service. Nearly one-half the series was studied both before and after operation. The patients were divided into twelve groups. The tests were made with the 1 *A* audiometer. In the first group of persons with frontal tumors, the composite curves showed a tendency to fall off at 2,048 double vibrations, while the bone conduction seemed to be somewhat lengthened at the lower levels, and distinctly so at 512 double vibrations. The Weber test was not lateralized. In the group of persons with pituitary tumors, the air and bone conduction was about the same as in those with frontal tumors, except for the lengthening of the bone conduction at about 256 double vibrations. The group of patients with parietal tumors showed a tendency of the air conduction to be generally lower throughout, and the bone conduction apparently lengthened throughout. The group of persons with occipital tumors showed a slight rise at 1,024 double vibrations and a direct drop throughout the rest of the scale in air conduction, and the bone conduction showed a general tendency upward except between 512 and 1,024 double vibrations. In the group of persons with extracerebellar tumors, acoustic neuromas and pontile angle tumors, air conduction was greatly depressed throughout the scale, more so on the side of the tumor, except in the pontile angle tumors. The bone conduction curve showed lengthening. The air and bone conduction curves tended to parallel one another. All these patients showed positive caloric reactions, deafness and vertigo. The Weber test was lateralized to the deafened side. Throughout the groups the left ear seemed to be more keen both by air and bone conduction than the right ear. In the group with intracerebellar tumors, the composite air conduction curve showed a tendency to upward bowing, the peak being at 1,024 double vibrations. The bone conduction curve bent downward, the peak being at 512 double vibrations, with a slight lengthening throughout. The patients with arachnoiditis showed

7. Drury, D. W.: Aural Acuity and Brain Lesions: 1. Audiometric Studies, Ann. Otol., Rhin. & Laryng. **40**:682 (Sept.) 1931.

a peaked curve upward at 1,024 double vibrations, the upper level of the low end being somewhat depressed. The bone conduction curve showed a tendency to a flatness from 256 to 4,096 double vibrations with an upward tendency toward normal at 8,192 double vibrations, with a lengthening in time throughout. The group of patients with multiple sclerosis showed a sharp drop at 2,048 double vibrations in the air curve and the bone conduction lengthened throughout but slightly at the lower frequencies. In a group of patients with intracranial tumors there was a general tendency for a slight rise at 1,024 double vibrations, and then a distinct drop to the end of the scale in the air conduction. The bone conduction showed a tendency toward normal up to 512 double vibrations and then a regular lengthening of time to the end of the instrument.

The most striking single feature of this study was the demonstration of deafness by objective means without a history of its presence. The explanation for this is that the defect was in the upper tones superior to the speech area. When the air and bone conduction curves approached the normal, there was a striking paucity of definite neurologic signs in addition to negative findings in the brain. In the entire group of patients, excluding those with acoustic neuroma tumors, only 0.03 per cent showed any degree of deafness or tinnitus. As a class of tumors, those of the frontal area seem to show the least effect on the ear. Careful audiometric study may prove to be of aid in the diagnosis and localization of brain lesions.

Heermann [8] called attention to the fact that Bloch, in 1893, suggested using two similarly pitched tuning forks, one at each ear, in order to determine whether a patient had some remains of hearing in one or the other ear. This depended on the fact that when one of the forks is brought closer to, and again taken away, from one of the ears, there is a marked increase in the intensity in the better hearing ear, or the patient states that he hears the sound in the head itself. Heermann also cited Stenger's description in 1900 of his now famous test, to the effect that when one fork is approached to the better hearing ear, that is, the normal ear, the sound can be definitely heard and the distance noted. Then a similarly pitched fork is brought before the supposedly deaf ear, and the fact noted as to whether the tone of the fork near the well ear masks the sound going to the supposedly deaf ear or whether it is necessary to approach the supposedly good ear still more in order to mask the sound entering the supposedly deaf ear. In other words, Bloch suggested that the first fork be held quietly close to the well ear and the second fork gradually approached to the supposedly diseased

8. Heermann, H.: An Important Source of Error in the Bloch-Stenger Test, Ztschr. f. Laryng., Rhin., Otol. **22**:423 (June) 1932.

ear and the patient asked whether he hears the tone of the first fork, namely, that one at the well ear, louder. Stenger, on the other hand, holds the fork quietly close to the supposedly deaf ear and then approaches the well ear with the other fork and asks the patient whether he hears anything. Heermann stated that when a simulator has been properly coached he can tell when the fork has been moved back and forth by the increase or diminution of the intensity or that he knows which way the fork is being held. For this reason, the original method of Bloch is considered better than that of Stenger.

One of us (Dr. Sonnenschein) has discussed the Stenger test on several occasions in these reviews, feeling that it is the most satisfactory for discovering simulation of total unilateral deafness. When the patient merely claims some impairment of hearing but not a total loss, the test is of practically no value. When desiring to evade army service or when endeavoring to collect insurance for injury, the malingerer usually states that he is completely deaf in one ear. Two forks of the same pitch (as a rule, two *a* 1, 435 double vibrations) are used. If, for instance, the patient pretends to be totally deaf in the left ear, the procedure is as follows: He is blindfolded (and told that he can concentrate better if his eyes are covered) and the vibrating fork is brought closer and closer to the right ear (the well ear) until he admits hearing it, let us say, at 20 cm. The fork is then brought closer and closer to the left or the supposedly deaf ear; of course, the person who is actually deaf will say he hears nothing, but if he is simulating, he will say he hears nothing in that ear. Then both forks are struck and the first one held 20 cm. from the right or well ear, at which distance he admitted hearing, and the other fork is brought gradually closer to the left or supposedly deaf ear. If the person is actually deaf in his left ear, the sound of the fork at that ear will not be heard but he will still hear the fork which is vibrating at a distance of 20 cm. from his right or well ear. If, on the other hand, he is simulating, when the fork is brought closer than 20 cm. to his left ear, the sound going into that ear will be much louder than that going into the admittedly well right ear (for the intensity of sound varies inversely as the square of the distance) and he will therefore not hear the fork which is sounding at a distance of 20 cm. from the well (right) ear. He cannot admit that he hears it in the left ear because he has claimed deafness in that ear. He does not know that the other fork is still sounding at a distance of 20 cm. from the right ear, but is masked by the intense sound entering the left ear. He therefore says he does not hear anything at all and is thus trapped. One can verify this masking of sound in a perfectly normal patient or colleague, for when blindfolded, if one fork is held at a distance of 20 cm. from the right ear and another sounding fork at a distance of, let us say,

10 cm. from the left ear, the person will hear the sound only in the left ear. Of course, he may guess the fact that two forks are being used and will say he hears it is in his right ear, but as a matter of fact, he only hears it in the left, because the intensity of sound entering that ear is so much greater than that entering the right ear.

We believe, therefore, that Heermann's objection to the Stenger test is not very important; if the fork is held still before the ear instead of moving it back and forth, as in the original Bloch procedure, there will be no difficulty in detecting simulated unilateral deafness.

Fabricant and Sommer [9] have two articles, one written in German, published in *Monatschrift für Ohrenheilkunde und Laryngo-Rhinologie,* and the other in English, published in the ARCHIVES OF OTOLARYNGOLOGY, which discuss the significance of the movements of the auricle when the hearing is tested by medium high pitched tuning forks. The authors stated:

> That the auricles of both human beings and animals show involuntary movement on acoustic irritation has been established in otologic literature since the beginning of the last century. This is known as Preyer's reflex as it was he who revealed that the striking of tuning forks caused an involuntary jerking and reflex of the auricle in new-born guinea-pigs and young rats, and this phenomenon has been noted time and again and has been constantly employed in physiologic experimentation on animals as a proof that the animal hears.

Reflex movements have also been noted in man, among others by Lincke, Politzer, Urbantschitsch and Lucae. The authors stated that they had been unable to find references either in English or in German to articles regarding a practical clinical utilization of the reflex movement in man.

They made tests on one hundred and ninety-eight unselected patients, medium and high pitched forks being used. Among these were patients with normal hearing with disease of the upper respiratory tract but negative findings in the ears, patients with external otitis, collection of cerumen, chronic catarrh of the middle ear, acute and chronic otitis media, former simple and radical mastoid operations, cochlear and vestibular lesions, deafness, deaf-mutism and otosclerosis. In fifty-five the reflex was found to be positive. In these there were either cochlear or vestibular lesions, or involvement of the middle ear with secondary lesions in the inner ear. The authors concluded that in response to tuning forks, the auricular reflex is not rare and has some diagnostic significance. It consists in a rhythmic jerking of a part of the whole of the auricle, in and out and up and down. Sometimes the reaction is

9. Fabricant, N. D., and Sommer, Ignaz: Diagnostic Significance of Auricular Reflex, Monatschr. f. Ohrenh. **66**:972 (Aug.) 1932; A New Test for the Diagnosis of Disease of the Inner Ear, Arch. Otolaryng. **16**:360 (Sept.) 1932.

only of the helix, at times of the lobule of the auricle. The most marked movement was obtained with medium and high pitched forks. The auricular reflex is obtained by holding the tuning fork ad concham or placing it over the patient's forehead or mastoid prominence. The operator must be careful to do this by bright daylight so as not to have any possibly misleading shadows.

One must also guard against confusing the reflex movements with those movements of the auricle so often noted in breathing, pulsation of the veins of the neck, chewing and voluntary facial movements. The authors stated that the cases in which the reflex was positive there was usually an infection of the inner ear and most frequently a combination of involvement of the middle and inner ear. No movement was noted in cases of otosclerosis. The authors believe that their findings show that the auricular reflex may be used for clinical diagnosis. If the findings are positive, involvement of the cochlear nerve is suspected, more often primary than secondary, but if the reflex is absent, a lesion of the inner ear cannot be excluded. They feel that the reflex appears in cases of slowly progressive lesions of the inner ear. They believe these patients listen more attentively than those with normal hearing, and that the added effort produces a more intensive tone in the muscles of the auricle.

The observations of the authors are interesting from a clinical standpoint, but it is too early to state whether this test will be of any real importance in diagnosis of hearing defects. Differentiation between the inner ear and middle ear or combined lesions can usually be made by the voice tests and tuning fork tests.

Becker [10] presented certain basic propositions and experiments regarding perception of sound by bone, cartilage and soft tissue. A vibrating fork in contact with the skull will impart the vibrations a variable distance in the surrounding air. This he proves by having one place his ear close to, but not in contact with, the activated skull, whereby the sound will be heard faintly by the observer but not at all when the fork is taken off the skull. In addition, the vibrations of the skull activated by the tuning fork are imparted to the air contained in all the skull cavities. This is proved by taking a rubber tube with eartips and placing one tip in the observer's ear and the other in the patient's ear, nose or mouth, and the sound will be distinctly heard. These vibrations spread in all directions but are heard loudest and longest at a zone opposite the point of contact of the fork and skull. For example, if the fork is held over the left mastoid the vibrations are heard best over the right mastoid or if placed on the vertex, they

10. Becker, B. M.: Perception of Sound by Bone, Cartilage and Soft Tissue Condition, Eye, Ear, Nose & Throat Monthly **11**:306 (Sept.) 1932.

will be heard best from the chin. This can be definitely ascertained when a stethoscope is applied to various parts of the patient's skull. The intensity of these vibrations emanating from the cavities of the skull is in direct ratio to the size of the cavity, being louder from the buccal cavity than the nose and louder from the nose than the ear. These same air vibrations in the cavities of the skull are also in direct ratio to the degree of closure or obstruction of other openings.

Becker expressed the belief that sound propagation through bone takes place in three ways, namely, by direct transmission of air, craniopneumatic conduction, and by direct excitation of the tympanic structures and the obturators of the labyrinthine windows, craniotympanic conduction. In the first instance, the air vibrations caused by the vibrating fork placed on the skull and the vibrating skull itself enter the external auditory meatus. In the craniopneumatic type, the vibrations set up in the cavities of the skull enter the labyrinth via the tympanic structures and the two windows, and if the tympanic structures are functionless, the vibrations are transmitted directly through the windows. In craniotympanic conduction, the resonating cranium sets into vibration also the tympanic membrane, ossicles and obturators of the foramen ovale and foramen rotundum and enter the inner ear through the two labyrinthine openings. The most important of these is the craniopneumatic route.

Cartilage conduction is produced by a vibrating fork placed on the cartilage of the external ear and takes place first on direct air conduction and secondly through chondropneumatic conduction. Cartilage conduction is better than bone conduction, especially from the tragus, which is in more intimate contact with the skull than other areas of the auricle. This was described by Stefanini, Lewis, Sonnenschein and others. The cartilaginous ear is an excellent resonator. When a vibrating fork is placed on the auricle, the air in the external canal and tympanum as well as the external air in the immediate vicinity of the auricle is activated to a much greater degree than in bone conduction, and is carried to the inner ear by way of the tympanic structures. The vibrations induced in the skull are too feeble to activate the tympanic structures. The excitation for the inner air as in bone conduction is the chief contributory factor in chondral conduction. The intensity of the sound from the tragus is in direct proportion to the inward pressure exerted on it. This also applies to the auricle when it is folded over to occlude the external meatus and a tuning fork is firmly applied to the back of it.

The sound emanating from the soft tissues when activated by a strongly vibrating fork is rather feeble and short lived, because these tissues absorb the sound and are poor sound conductors. The intensity is greatest from the lobule of the ear, less from the cheek and least from the tip of the tongue. The vibrations from the lobule and the cheek enter

the inner ear the same as those from the cartilage, namely, the combined direct and indirect air conduction. The vibrations from the tongue enter the labyrinth only through indirect air conduction. The vibrations from the soft parts are transmitted to the cranium, and can be heard when placing the ear firmly against the skull at a point approximately opposite to the point of application of the fork.

The author has carried out his experiments systematically and industriously.

Becker [11] tested different parts of the skull for their capacity to transmit sound under normal conditions of hearing. He found that the teeth of the upper maxilla, especially over the central incisors, are the site of maximum conductivity for both intensity and duration. In addition, forks from 32 to 256 double vibrations inclusive will be heard better with the teeth of the upper and lower jaws separated, whereas the forks from 512 to 2,048 double vibrations inclusive will be heard better with the teeth in contact. The second most favorable site is shared equally by two separate zones on the temporal bone. For forks of middle and high range, the region of the mastoid antrum is best, and for those of the low pitch the area of the squama directly in front of the ear and above the root of the zygoma is more favorable. The site of minimal conductivity for all frequencies is shared about equally between the glabella and the inion and the adjacent vicinities in a vertical direction.

Becker expressed the belief that bone conduction is a modified or indirect form of air conduction. The vibrations produced when the skull is activated do not directly excite the intralabyrinthine structures. They enter the inner ear in two ways when the tympanic structures are intact and functioning. The first is by craniopneumotympanic conduction, which is an activation of the air in the external and middle ears, which in turn produces vibrations of the drum membrane, and is then transmitted by the ossicular chain to the fenestrae and the inner ear. The second is by craniotympanic conduction, where the bones of the skull directly transmit their vibrations to the tympanic membrane, and ossicles, which in turn transmit them to the inner ear by way of the two windows. When the drum and ossicles are not present, the vibrating air of the outer and middle ears transmit their vibrations directly to the inner ear by way of the oval and round windows. From this, one sees that the chief factor concerned in the process of hearing by bone conduction is the agitated air in the meatus and tympanum.

Becker emphasized the point that conductive and obstructive deafness are not the same thing. Conductive deafness, he said, should only be

11. Becker, B. M.: Zones of the Skull of Maximal and Minimal Sound Propagation: Hearing Through Skull and Hearing Through Telephone, Laryngoscope **42**:588 (Aug.) 1932.

used for lesions affecting and confined to the intratympanic structures which interfere with audition, as the drum membrane, ossicles and the two windows. Obstructive deafness should be applied for conditions confined to the external ear which impair the hearing either by partially or by totally occluding the external meatus. This also applies to occlusions of the eustachian tube. In obstructive conditions, the impairment of hearing and prolonged bone conduction bear a definite relationship to the degree of obstruction. The greater the obstruction, the greater the loss of hearing and prolonged bone conduction. The prolonged bone conduction in obstructive conditions of the meatus and auditory tubes is a true phenomenon and lends itself to a logical explanation. However, this same phenomenon in the conductive impairment is only an apparent one and its cause is not known.

Becker also showed that the degree of sound perception by bone conduction in artificial obstructions depends directly on the size of the closed cavity. With the finger placed tightly in the external canal, the sound is heard weaker than when the finger is placed lightly in the canal. This latter in turn is weaker than when the palm of the hand is placed against the ear producing the loudest sound because the meatal space is greatest. The maximum expression for prolonged bone conduction can be obtained by placing a 512 double vibration fork against the teeth. This procedure will give a negative Rinne test even in normal conditions of hearing. He also showed that persons with conduction impairment hear better over the telephone because they convert the ear canal into a closed cavity by pressing the receiver against the deafened ear. In this way the vocal vibrations poured into this cavity are capable of exciting the confined air in the closed cavity to greater activity than is possible in the open canal.

Becker [12] again presented his views and experiments on bone conduction in the ARCHIVES similar to those in his paper in the *Ear, Nose and Throat Monthly* of September, 1932. Here the experiments are carried out first on a dried skull and then on the living. One observation seems to differ somewhat in the two papers. In this article, he stated "that when a vibrating fork is set on the skull, the sound whether by ear or through an instrument will be heard best at or near the site of application of the fork and the next best place will be at a point opposite the zone of contact—cross or diagonal conduction." In his other paper he stated that these vibrations spread in all directions but are heard loudest and longest at a zone opposite the point of contact between the fork and bone. This difference may be due to observations made in the first instance on the dried skull and in the other on the living subject.

12. Becker, B. M.: Hearing by Bone Conduction, Arch. Otolaryng. **16**:364 (Sept.) 1932.

He again stated that "sounds perceived by bone conduction enter the inner ear via the external ear and the middle ear with its component structures if these are intact, or they enter the internal ear directly through the two fenestrae if the drum and ossicles are destroyed." He concluded that bone conduction is a modified form of air conduction.

Moose,[13] in using an apparatus for activating tuning forks that would give standard blows of different intensities for the purpose of accuracy and to save time, found that such a device is not necessary. In a series of tests in which the tuning fork was activated from eight different positions, he discovered that strong blows delivered in any manner desired always gave uniform results. However, there is a great difference between a weak and a moderately hard blow. These conclusions verify the remarks of both Randall and, more recently, of one of us (Sonnenschein), that a satisfactory method of excitation is to hold a fork vertically and let it drop of its own weight to the cushion of the knee.

A comparison of the calculated and actual differences between the blows shows that a tuning fork exciter is not necessary. There are other sources of error which should be considered. The first is the distance the fork is held from the ear. Moose found a definite difference when the *c* 1 fork was held first a half inch and then a quarter of an inch away from the ear. There is actually a hearing loss of 10.9 decibels, or 9.8 per cent, when the test is made at a half inch away from the ear instead of one-fourth inch. This is not so for frequencies of 4,096 and 2,048 where the difference is only slightly if at all appreciated. The second source of error is the environment in which the test is made. Knudsen and Jones showed that the "normal ear exhibits between 12 and 15 per cent better hearing in the sound proof room. Also, that patients with fixation of the stapes, etc., who show increased bone conduction in the office do not show it when tested in a sound proof room. Therefore one has been testing the degree of noise in the room rather than the cochlea of the patient." Moose by his tests showed that the noise in one of his outside office rooms amounts to 20 decibels, which is capable of reducing the acuity of hearing of a normal person 18 per cent. That means a patient could have a loss of hearing of 18 per cent without its being detected. His conclusions are that tuning forks may be activated by any means desired provided a strong blow, which might be called a standard blow, is used. A source of error may lie in holding a *c* 1 fork at variable distances from the ear, and a sound proof room is necessary for both diagnosis and prognosis. He added also that unless each tuning fork has its damping constant determined the findings cannot be properly interpreted.

13. Moose, R. N.: Tuning Fork Exciter, Arch. Otolaryng. **15**:557 (April) 1932.

Roth [14] presented a method and chart for recording the hearing acuity for air conducted sounds produced by the tuning forks. The advantages of this method over the audiometer are the absence of a mixture of bone and air conduction, portability and inexpensiveness. The principles of this method are based on several facts. The greatest intensity of sound occurs with the striking of the fork. The period from the time the fork is struck to the point of lost audibility in the normal ear is called the period of diminishing vibration. There is a constant ratio between the force of the blow, the intensity of the sound and the length of the period of diminishing vibrations. As a result, the intensity may be measured in terms of seconds from any particular point of intensity or loudness that one may wish to record. The point of intensity is the last audible sound heard by the patient. The interval from this point to the last audibility for the fork for the normal ear will be termed the acoustic differential. A record and chart for each of a series of forks can be made by this method which will show the hearing acuity of the patient as compared with the normal ear.

A separate graph is used for each ear, with space for a record of both before and after inflation. The test is made by striking the fork with moderate force and holding it a half inch away from the patient's ear. When the patient states he no longer hears the sound, the fork is held to the examiner's ear, providing his hearing is normal, and the time is noted from then until the point of lost audibility for the normal ear. The number of seconds is then charted. This is repeated for the remaining forks, and the resulting line is called the acumetric line. The examiner familiar with the time limits of the various forks can readily evaluate the extent of loss of hearing of the patient. Instead of charting such a line, Roth has determined the vibrations period required to produce perception of sound in the almost totally deaf ear. This was experimentally determined by testing patients totally or almost totally deaf, and is represented on all the charts as the borderline of total deafness. This is helpful in the interpretation of the results and determination of the approximate field of residual hearing. In the patient who is extremely hard of hearing when a fork is heard for only a few seconds, this can be charted by counting upward from the borderline of total deafness, thereby saving considerable time. Copies of these charts are presented in Roth's article.

We wish to call attention to the fact that a moderate difference in the force with which a fork is excited makes no difference in its rate of decrement. If, however, a fork is struck with very great force one time and only lightly another, then there will be a definite variation in the duration of the vibration.

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14. Roth, Aaron: A Graphic Method and Chart for Hearing Tests with Tuning Forks, Arch. Otolaryng. **15**:834 (June) 1932.

In order to explain the occasional finding of a shorter bone conduction over one mastoid than the other when the Weber test was lateralized to the former, Mackenzie [15] began investigating this Weber-Schwabach paradox. In cases of shortened bone conduction, as in perceptive deafness, or prolonged bone conduction in obstructive deafness, the change in the bone conduction is noticed more over the mastoid on the affected side, less over the midline of the skull and least over the unaffected mastoid. This occurs so consistently as to be regarded as a working law. However, the exceptions, although occasional, do occur, and investigation disclosed it to be present consistently in cases of mastoiditis, and in no other form of obstructive deafness. Mackenzie found "no case of mastoid disease in which the fork was not heard more poorly on the affected mastoid than it is supposed to be heard, judging from the strength of lateralization of the Weber to that side, and the increased length of the bone conduction in the midline and on the opposite mastoid." The possible explanation, he believes, is that a disintegrating mastoid acts less favorably as a conductor of sound waves when the fork is applied to it than when held over a perfectly intact mastoid. Mackenzie presented three cases showing the three possible degrees of the Weber-Schwabach paradox. The first degree is where the bone conduction in the affected mastoid is lengthened over the normal, but not sufficient in view of the strong lateralization of the Weber test to that side and the lengthening of the bone conduction in the midline. The second degree is where the bone conduction over the affected mastoid is neither lengthened or shortened, and the third degree is where the bone conduction over the affected side is shortened with an increase over the midline and on the opposite side. All these occur in cases of suppurative middle ears, and Mackenzie expressed the belief that they represent the three stages of mastoiditis. However, a second degree paradox may be found in a first stage of mastoiditis and likewise a third degree in a second stage. This may be due to the sensitiveness of the test and the fact that the stages of mastoiditis are not so sharply defined. It also may be possible to find a second stage of mastoiditis present in one part of the mastoid and a third stage in another. He stated that up to this time the Weber-Schwabach paradox has proved to be 100 per cent accurate both in a positive and in a negative way. He has found no case of positive mastoiditis in which the test was negative, nor a positive test where there was an absence of mastoiditis. The test is of value especially in those cases in which the findings of mastoiditis are not definite. This test cannot be made with the audiometer.

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15. Mackenzie, G. W.: More About the Weber-Schwabach Paradox, Eye, Ear, Nose & Throat Monthly **11**:225 (July) 1932.

The purpose of the Weber test is for lateralization, so that speaking of a "shortened" Weber test as the author does is a little misleading (R. S.).

Jenkins [16] discussed paracusis willisii and otosclerosis, especially that type which has been confused with middle ear deafness. In cases of otosclerosis, the conversational voice and whisper are heard at about the same distance, whereas in the middle ear deafness the whisper is heard at a much shorter distance than the conversational voice. He stated that many otosclerotic patients remark that they hear a voice but cannot make out the words. On testing these patients in a large room, there was a difference of over 30 feet between the distances at which the voice was heard and words recognized. In this same room the normal person heard the whispered voice at 36 feet. Patients with otosclerosis often notice certain bone conducted noises as in mastication or walking on hard pavements, whereas in middle ear deafness they do not observe this. The otosclerotic patient hears better over the phone than in ordinary conversation, which is not true in middle ear deafness. Tinnitus is often the first symptom noticed in otosclerosis and is frequently like a musical tone. When it is of this type and patients state that they would rather have the noise than absolute quiet even when they are stone deaf, Jenkins considered that it is almost diagnostic of otosclerosis. A tinnitus of this sort in middle ear disease is an indication that the labyrinth has been somewhat affected.

The lower tone limit is comparatively higher in cases of otosclerosis than in cases of middle ear deafness. The tone limit of a negative Rinne test is the highest tone at which the Rinne test is negative. This may be as low as 90 double vibrations in patients with only a slight degree of deafness. Below this, the test is unreliable. The tone limit becomes higher as the deafness increases, reaching as high as *c* 3 in very deaf patients. In two patients who hear conversational voice the same, the lower tone limit and the tone limit of the negative Rinne test are higher in the person with otosclerosis than in the one with middle ear deafness. There is no marked difference as regards high tones except in advanced cases in which the upper tone limit may be very low in otosclerosis. The upper tone limit is usually higher by bone than by air conduction. In early cases of otosclerosis a slight loss in hearing for low tones by bone conduction is often found, but never in pure cases of middle ear deafness. Patients with otosclerosis appreciate the differences in the Weber and Rinne tests more definitely than do those with middle ear disease. These are the essential differences between the otosclerotic syndrome and middle ear deafness.

16. Jenkins, G. J.: Paracusis Willisii and Some Clinical Features of the Otosclerosis Syndrome, J. Laryng. & Otol. **47**:517 (Aug.) 1932.

The clinical features that play some part in the phenomenon of paracusis are the raising of the low tone limit, negative Rinne test and usually some loss of bone conduction. The diminution in acuity is much more marked in the low tones than the high tones, and as a result the persons with otosclerosis hear women's voices better than men's. Noise helps the otosclerotic patient by paracusis and by giving relief from the tinnitus. The two elements in the noise which are important factors in paracusis are the pitch of the tone and the volume of sound. The noise must be comparatively low toned, and volume will vary from a soft to a thundering noise. There is a similar alteration in the conversational voice with variations in the volume of the confusing noise. The quieter the room the softer the voice and the lower the tone, and vice versa. If the normal speaker passes from a quiet to a nosiy place, he increases the volume and raises the pitch of his voice. These are important factors in the symptoms of paracusis. The otosclerotic syndrome converts a noisy place, especially when it is due to a low rumbling noise, to an almost quiet one, and the hearing is helped by the increased volume and higher tone of the voice. The symptoms of paracusis are possible only with the deafness syndrome found in most cases of otosclerosis. Paracusis is sometimes found with deafness of osteogenesis imperfecta, osteitis deformans, and occasionally chronic suppurative middle ear disease. In osteitis deformans, the diminished acuity of hearing by bone conduction is usually greater than in otosclerosis, so the syndrome is more like that of otosclerosis with an internal ear impairment more marked than usual. In osteogenesis imperfecta, as shown in Nager's case, there are true otosclerotic bone changes which produce the syndrome rather than the general disease. Paracusis can only occur when the hearing syndrome found in many cases of otosclerosis is present.

As far back as 1862, von Troeltsch stated that the phenomenon of paracusis willisii rests on the fact that in a very noisy environment the person with normal hearing raises his voice, so that the otosclerotic patient hears better, partly because of this increase in the loudness of the normal hearing speaker, and partly because he is not disturbed by low pitched noises about him because of his hearing impairment for the lower octaves.

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Abstracts from Current Literature

Ear

DIAGNOSIS AND TREATMENT OF ACUTE OTITIS MEDIA. H. B. FORSTER, Brit. M. J. **2**:645 (Oct. 10) 1931.

Acute otitis media is discussed, special stress being placed on the etiology rather than on the specific bacteriology. Heredity, according to the author, plays a part, as do also congenital defects and atrophic disease of the nose and accessory sinuses. He states finally that the lymphadenoid tissue of the upper respiratory tract has been maligned unnecessarily. Clinical signs are given, as well as indications for incision and after-treatment. Special attention is paid by the author to the differential diagnosis of acute otitis media from other causes of acute earache.

ROYSTER, University, Va. [AM. J. DIS. CHILD.]

LATERAL SINUS THROMBOSIS WITH METASTATIC INFECTIONS OF HIP JOINT. DOUGLAS GUTHRIE, Tr. Med.-Chir. Soc., Edinburgh, 1932, p. 13; in Edinburgh M. J., February, 1932.

Several months following otitis of scarlet fever in a girl 9 years of age, there appeared headache, vomiting, fever and a rigor, without tenderness of the mastoid. These symptoms were due to the presence of sinus thrombosis, which was discovered at operation. Subsequent spreading of the infection required further operative procedures. Evidently metastasis from the focus in the ear reached the hip, as arthritis then developed. Removal of the head of the femur was followed by ankylosis of the joint and, after several months, recovery.

NEFF, Kansas City, Mo. [AM. J. DIS. CHILD.]

MÉNIÈRE'S DISEASE AND MECHANICAL AND PSYCHIC TRAUMA. J. KOCH, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:29 (July 23) 1932.

The explanation of clinical pictures resembling Ménière's disease by examinations of the ear and equilibrium tests alone is not possible. A careful examination of the entire organism is necessary. Every attack of vertigo that is accompanied by spontaneous symptoms and deviations of the experimental excitability of the vestibular apparatus is provoked by disturbances of the vestibular ocular reflex mechanism. These may be based on organic and neurotic vascular changes. Weakness of the vasomotor system is often constitutional, but it may also be provoked by mechanical trauma of the skull. It is doubtful whether hysteria as such can cause vertigo with spontaneous symptoms and experimentally demonstrable vestibular disturbances. The author's observations of disturbances of the labyrinth following psychic traumas indicate that these disturbances are based on vasoneurotic changes.

EDITOR'S ABSTRACT.

PUNCTURE OF THE MIDDLE EAR: DIAGNOSTIC SIGNIFICANCE. A. REINIGER, Monatschr. f. Ohrenh. **66**:694 (June) 1932.

Reiniger thinks that puncture of the middle ear and bacteriologic examination of the fluid aspirated are a valuable aid in the diagnosis of atypical otitis media. The atypical case may, soon after a severe onset, show great improvement with a tendency to spontaneous healing which, however, is arrested after a while, the tympanum remaining inflamed and the auditory acuity decreased. In these cases bacteriologic examination of the secretion of the middle ear may help to determine

whether the slow healing is due to a disturbance in pneumatization, to general disease or to mucosus otitis, presumably requiring an operation. In cases which from the beginning lack characteristic symptoms and in which the subjective symptoms appear harmless, severe symptoms, such as headache, high temperature or vertigo, may suddenly supervene. Puncture of the ear may help to determine whether these threatening symptoms are due to otogenous complications or intercurrent disease bearing no relation to the ear. In other cases the subjective symptoms, slight from the start, may continue unchanged for weeks, not responding to conservative therapy, while the appearance of the tympanum is more that of catarrh of the middle ear than of acute otitis. Here again bacteriologic examination may determine the diagnosis. The author recommends puncture with a long needle and aspiration of the fluid with a 1 cc. syringe. This enables one to determine immediately the existence of secretion and note its appearance. The fluid aspirated is tranferred to broth Gram stains and Voges agar plates are made from the broth suspension. The author cites several cases in which this method helped to establish the diagnosis.

EDITOR'S ABSTRACT.

MUCK'S EPINEPHRINE PROBE TEST IN DEAF-MUTES. A KÖRÖSI, Monatschr. f. Ohrenh. **66**:942 (Aug.) 1932.

Körösi found the use of Muck's epinephrine probe test a valuable aid in establishing the etiology of deaf-mutism. Determination of the etiology of deaf-mutism is of interest not only from the standpoint of science, but from that of instruction, as deaf-mutes with remnants of hearing (mostly the congenital deaf-mutes) do not belong in institutions for deaf-mutes but in schools for persons with bradyecoia. The anamnesis is often unreliable in establishing the etiology, because of the unwillingness or inability of parents to give the true facts. The author tried the epinephrine probe test on 116 deaf-mutes. The test subjects were grouped according to Alexander and Fischer's classification of deaf-mutes, which is based on the somatic conditions of the person and his family and the basic etiologic symptoms revealed by anamnesis and clinical examination. The epinephrine probe test was negative in hereditary degenerative disease of the labyrinth. It was negative in patients with individually acquired labyrinthine disease due to intra-uterine trauma or infection. It was also negative in normal persons and in persons of constitutional inferiority who acquired the labyrinthine disturbance after intercurrent disease, either acute or chronic infections. A positive reaction, the white streak sign, was obtained in normal patients or patients of constitutional inferiority in whom labyrinthine disease appeared following trauma or meningitis. The results agreed with the author's anticipations in all but three cases.

EDITOR'S ABSTRACT.

DIAGNOSTIC SIGNIFICANCE OF REFLEX OF PINNA. N. D. FABRICANT and I. SOMMER, Monatschr. f. Ohrenh. **66**:972 (Aug.) 1932.

Fabricant and Sommer noted the occurrence of involuntary movements of the pinna in response to sound stimuli in 55 of 198 nonselected patients, consisting of persons with normal hearing and persons with various forms of disease of the ear. The movement was produced by holding a tuning fork in front of the ear or over the mastoid process. It consisted in a rhythmic twitching of a part of the pinna and outward and inward movement of the whole auricle, or, occasionally, movement of the helix or lobe only; an up and down motion was rare. The movements varied in amplitude from a barely discernible movement to one readily seen. Usually they occurred with the use of weak tones; loud tones provoked no movement. Concurrent movement of other muscles was not observed. In almost all of the cases in which the reflex occurred, disease of the labyrinth existed. More rarely, the middle ear and the labyrinth were both diseased. In only two of the cases with a positive reflex was the labyrinth free from disease; one was a case of pure otitis media and in one the ear was normal. The move-

ment is more pronounced in pure labyrinthine disease than in combined disease of the middle and the inner ear. In general, it is the more pronounced the greater the decrease in auditory acuity. The authors conclude that the reflex of the pinna has diagnostic value. A positive reflex indicates a strong probability of injury to the auditory nerve, usually primary, occasionally secondary, as in disease of the middle ear. However, absence of the reflex does not exclude injury of the auditory nerve. The author thinks this reflex may be due to an increased receptivity of the cerebral cortex and a heightened irritability of the cortical sphere of hearing resulting from the effort to hear which is made by the person with impaired hearing. This straining to hear also produces an increased tension in the muscles of the ear.

EDITOR'S ABSTRACT.

OTOGENOUS EXTERNAL PACHYMENINGITIS. L. Z. FISHMAN, Monatschr. f. Ohrenh. **66**:981 (Aug.) 1932.

Fishman states that when the focus of external pachymeningitis has been exposed the surgeon is frequently confronted by the question as to whether all diseased parts have been exposed or whether intervention should extend to this focus. This question is justified by the fact that extradural abscesses, especially those of the middle cranial fossa, may simulate an abscess of the temporal lobe in practically all of its symptoms. Extradural abscesses of the posterior cranial fossa occasionally imitate a sinus thrombosis. The author illustrates his points with several case reports chosen from a study of twenty-eight cases of external pachymeningitis and discusses the symptomatology. In pachymeningitis of the middle cranial fossa, there is headache on the diseased side, but it is not localized with any frequency in a certain section of the skull. Labyrinthine vertigo is frequently found. It is attributed to pressure on the cerebellum. In pachymeningitis of the middle cranial fossa there is frequently a history of chills, attributed by the author to involvement of the superior petrous sinus. The temperature frequently rises above 38.3 C. (101 F.) in pachymeningitis of the middle cranial fossa. In periphlebitis occurring in the course of an acute exacerbation of a chronic otitis, temperatures of 38.3 C. (101 F.) occur. If the temperature rises above this, disease of the sinus lumen should be suspected. Periphlebitis in acute or subacute otitis may reach temperatures up to 40 C. (104 F.).

EDITOR'S ABSTRACT.

DEEPLY RETRACTED PARS FLACCIDA AND CHOLESTEATOMA. W. LANGE, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:575 (July 22) 1932.

Lange says that in acute inflammations of the middle ear the proliferation of the epidermis of the external meatus into the connective tissue of the inflamed and thickened pars flaccida may, with continuation of the inflammation, lead directly to the formation of a cholesteatoma which spreads in the mucous membrane under the epithelium. In other cases, following healing of the acute inflammation, there is a deep retraction of the pars flaccida, a condition that may remain permanently. In such cases a cholesteatoma may develop if a renewed inflammation of the tympanic cavity or even of the auditory meatus occurs. If the retraction is very deep and narrow, the desquamated layers of the epidermis are not removed, or are removed only partially. In such cases a slow cystlike growth of the retracted part will occur even in the absence of inflammation, and a cholesteatoma will result. Inflammatory processes of a mild form are probably also present. They may lead rather early to a rarefying osteitis of the ossicles, in which the same condition is found which is present in the osseous wall of a cholesteatoma of the tympanic cavity and in the mastoid process. As the development of a cholesteatoma may in this way follow acute otitis media, it will readily be seen that a good pneumatization of the mastoid process may also be found with a developed cholesteatoma.

EDITOR'S ABSTRACT

Pharynx

POSTANGINAL SEPSIS. ISAAC A. ABT, J. Pediat. **1**:8 (July) 1932.

A survey of the literature on septicopyemia following tonsillar and pharyngeal infections is presented, with a clinical discussion of the atria of infection from the throat, and the pathology, etiology, clinical course, complications, prognosis and treatment of the condition. Four cases are reported briefly, and the frequency of thrombosis of the internal jugular vein is emphasized. The treatment recommended is mostly surgical, drainage of the septic focus and ligation of the jugular vein on the affected side if there are chills and irregular fever.

LEMASTER, Evanston, Ill. [AM. J. DIS. CHILD.]

RESULTS OF THE SO-CALLED TONSILS AND ADENOIDS OPERATION. JAMES KERR LOVE, Lancet **1**:1356 (June 25) 1932.

1. Adenoids should be removed early, and the best results are attained in the cure of mouth-breathing if the operation is followed by training of the patient to overcome the acquired habit.

2. Tonsils may often be left unless they are actually septic.

3. A discharge from the middle ear due to other causes, for example, scarlet fever and measles, is not removed by the operation on the tonsils or the post-nasal adenoids.

4. Nasal discharge is not always cured by such operations, but even in cases of apparent failure great improvement takes place.

These conclusions suggest to the author this thought: "We talk of the T and A operation: are we not the slaves of a phrase? Are we not dealing with two operations which are too often performed together? Should they not often be dissociated? The adenoid infection usually takes place earlier and the tonsils suffer first and very occasionally alone. I therefore plead for the removal of adenoids as soon as nasal obstruction occurs, leaving the tonsils unless they are evidently diseased. Where there is no history of sore throat and no enlarged glands I tend to leave the tonsils." The author finds that this view results in his leaving the tonsils in from 10 to 15 per cent of the patients sent to him for operation.

LANGMANN, New York. [AM. J. DIS. CHILD.]

ADENOIDS AND ADENOTOMY IN FIRST WEEKS AND MONTHS OF LIFE. H. NEUMANN, Monatschr. f. Ohrenh. **66**:922 (Aug.) 1932.

Neumann says that adenoid growths in infants who feed at the breast or on a bottle may obstruct nasal breathing to such a degree that sucking is interfered with and nutrition is disturbed. In the majority of these cases medical treatment will permit postponing adenotomy till the end of the first half year or year or at least beyond the first weeks or months of life, but in some cases surgical intervention becomes an immediate necessity. The author thinks that adenotomy is indicated if a palpable tumor filling the nasopharynx has been ascertained, if interference with feeding and nutrition results from the obstruction of nasal breathing, or if there is retarded development attributed to adenoids by the attending pediatrician. Three distinct types of tonsils occur in nurslings. The first is simple hyperplasia without accumulation of secretion. The second is irregular hypertrophy showing the beginning of the villous form frequently seen in older persons. This form usually has a heavy tenacious mucoid secretion and is often accompanied by nasal hypersecretion. The third form has a relatively small tumor, but heavy secretion in the nose and nasopharynx. Diagnosis in infants only a few weeks or months old is difficult. The author employs palpation with a sound introduced by mouth and indirect palpation with the finger to determine the resistance when the uvula is pressed back. The pressing down of the rear part of the tongue with a small laryngeal mirror frequently permits seeing the adenoid growths in the initial moment of the vomiting reflex. The author recommends

instillation of oil for one or two weeks before adenotomy whenever possible. He describes the technic of performing the operation with his modification of Beckman's curet. This curet possesses a prong which is pushed into the tumor, holding it firm. It is essential to use a sharp instrument and perform the operation in one stroke. An anesthetic is not used in children under 1 year of age. Hemorrhage is slight. The results of this intervention in nurslings are usually excellent.

EDITOR'S ABSTRACT.

OSTEOMYELITIS OF INFERIOR MAXILLA FOLLOWING TONSILLECTOMY AND CONSECUTIVE EXTRACTION OF WISDOM TOOTH. O. Voss, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **22**:474 (June) 1932.

Voss reports the case of a man, aged 23, in whom tonsillectomy was followed by suppuration of a wisdom tooth, succeeded by phlegmonous inflammation of the soft parts of the cheeks and throat and finally osteomyelitis of the inferior maxilla. The author thinks that if there was any relation between the tonsillectomy and the succeeding manifestations, it could have been only that the tonsillectomy had a sensitizing influence on the latent infection of the wisdom tooth. It is equally probable that it was merely an unfortunate accidental succession of the two processes. In any event, this case shows again the importance of scrupulous examination of the teeth and especially of a cutting wisdom tooth before tonsillectomy, so that all pathologic anomalies may be removed before the intervention.

EDITOR'S ABSTRACT.

Larynx

A CASE OF MULTIPLE PAPILLOMATA OF THE LARYNX WITH AERIAL METASTASES TO THE LUNGS. H. B. HITZ and E. OESTERLIN, Am. J. Path. **8**:333, 1932.

In a girl, aged 2, multiple papillomas of the vocal cords became detached and were carried into the bronchi by aspiration. They passed the larger bronchi, but were caught in the bronchioli, obstructing the lumens. In this way, they became implantation metastases and began to grow in the alveoli. No invasion of the lymphatics by tumor cells was observed.

[ARCH. PATH.]

NEW SUGGESTION FOR TREATMENT OF LARYNGEAL STENOSIS DUE TO BILATERAL MEDIAN FIXATION OF VOCAL CORDS. K. AMERSBACH, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:109 (Aug. 23) 1932.

Amersbach outlines his modification of Wittmaack's treatment of laryngeal stenosis due to bilateral median fixation of the vocal cords. The latter's method consists in downward transportation of one of the vocal cords. The author recommends moving the vocal cords downward in the region of the anterior commissure instead of in its posterior portion. After the performing of a laryngofissure the vocal cord which is to be transposed is anesthetized. A smooth cut is made through the ventricle of the larynx going all the way to the cartilage. Starting at this cut, the vocal cord, including the inner perichondrium, is detached from the cartilage until only a connection with the posterior part by means of the mucosa and surrounding tissue remains. The vocal cord can then be transposed downward to the median part of the conus elasticus, which has been slit at this point. Here it is firmly fixed with several sutures. It is important that the vocal cord be made sufficiently mobile that the transposition and fixation can be accomplished without tension. This method is simpler and less destructive than Réthi's or Wittmaack-Bahre's intervention on the arytenoid cartilage. Furthermore, since the arytenoid cartilage is not involved, and no musculature is destroyed, this method permits reestablishment of the original condition in case the function of the cords is restored.

EDITOR'S ABSTRACT.

CARCINOMA OF THE VOCAL CORDS. F. BLUMENFELD, Ztschr. f. Krebsforsch. **35**:360, 1932.

In a general discussion of the subject, Blumenfeld points out the great disproportionate incidence of carcinonoma of the vocal cords in males; the fact that it rarely causes lymphatic metastasis, and then usually in connection with involvement of the anterior portions of Poirier's glands and the absence of peculiarities as to age incidence. A peculiarity which he emphasizes is the existence of an early stage in which local removal is sufficient for permanent cure; this is true as long as infiltration is limited to the elastic tissues of the cord, while after infiltration into the muscular tissues, which is indicated by fixation of the cord or great restriction of its motility, this stage has been passed, and more radical operation is called for.

EGGERS, Omaha. [ARCH. PATH.]

LARYNGOSTROBOSCOPY. D. WEISS, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **22**:391 (June) 1932.

Weiss presents a comprehensive and critical study of laryngostroboscopy. In the first part he deals with the nature of stroboscopic images, the optical laws of stroboscopy, types of apparatus, technic of examination and sources of error. In the second part he deals with the physiology of the vibrations of the vocal cords, including participation of the vocal cords, the end-phase in the chest and falsetto registers, Rethi's wave, the direction of the vibration of the vocal cords and the shape of the vocal cords. The third part deals with the pathology of the vibrations of the vocal cords, including observations in laryngitis and paralysis of the recurrent nerves, alternating vibrations, stroboscopic standstill of one vocal cord, different vibration frequency of both vocal cords and other individual observations. The chief limitation of stroboscopy is that it is suitable only for strictly periodic vibrations, and even the singing voice can be regarded as such only with certain qualifications; for speaking, stroboscopy is actually inadequate. The holding of the tone by the test subject and comparison of the two tones by the investigator make musical demands on both. Exact information can be obtained only from observations in which the vocal cords vibrate slowly, which is sometimes hard to achieve. In rapid vibrations the subjective factors play an important rôle. This factor should be offset as much as possible by a strict systematic criticism of the stroboscopic image. Recognition of the special laws and limitations and the sources of error of this method should increase the value of its results.

EDITOR'S ABSTRACT.

Nose

ETIOLOGY OF THE SYNDROME OF THE NASAL NERVE. C. CHARLIN, Ann. d'ocul. **169**:257 (April) 1932.

The syndrome of the nasal nerve has been discussed by Charlin in two previous communications (*Ann. d'ocul.* **168**:86 [Feb.]; 808 [Oct.] 1931). In this paper he considers the etiology of the syndrome of the nasal nerve and points out that in the presence of the triad he has spoken of, that is, inflammation of the anterior segment of the eye, coryza with profuse watery nasal discharge and oculo-orbital neuralgia, examination should not be limited to exploration of the nasal cavity. He reports a case of the syndrome of the nasal nerve relieved of symptoms by the removal of several infected teeth. In the treatment of these conditions he first suggests applications of medicaments in the nose. If this does not relieve the condition in a few days, a more general cause should be sought. If a nasal pathologic condition or nasal deformities are found, these should be corrected.

BERENS, New York. [ARCH. NEUROL. & PSYCHIAT.]

CELLOPHANE NASAL TAMPONADE. H. NEUMANN, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:194 (Aug. 23) 1932.

Neumann recommends cellophane nasal tamponade for hemostasis following nasal operations and in epistaxis. One or several pledgets of gauze are wrapped in a piece of sterilized cellophane so that the gauze is contained in a sheath of cellophane closed at one end and increasing slightly in circumference toward the open end. The tampon is introduced into the nose, with the closed end first, until a slight resistance shows that it has penetrated the posterior nares. The protruding end is then cut off, and a strip of adhesive tape covered on the inside with a piece of cellophane is placed over the nasal orifice. Dry sterile gauze is used, but the cellophane, sterilized by boiling, is used wet. The cellophane has no absorbent effect, but, as it is merely in contact with the mucous membrane, not tightly pressed against it, the secretions and excess blood ooze downward along the cellophane where they are absorbed by the gauze at the open end of the tampon. The author has used this tamponade in eighty-five cases, twelve of them cases of epistaxis and the others of various nasal operations. He finds that it provides complete hemostasis with perfect asepsis. It is easily and rapidly introduced and can be removed rapidly and without irritation or injury of the mucous membrane, because it never adheres. It greatly reduces the usual discomfort of the patient and the posttamponade reaction. The author stresses the importance of making the tampon just the right size so that it is in contact with the mucosa everywhere without exerting pressure.

EDITOR'S ABSTRACT.

HARD PAPILLOMA OF THE NOSE. F. MOSSBÖCK, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:254 (Aug. 23) 1932.

Mossböck reports a case of hard papilloma of the nose in a man aged 70. The tumor filled not only the entire right nasal cavity but also the maxillary and ethmoid sinuses up to the sphenoid bone and had eroded the thin median bony wall. After radical extirpation, healing occurred without complications. The tumor was grayish red, the size of an apple and lobular and had a finely granulated surface. It belonged to the type of mixed columnar and squamous cell papillary epithelial tumor. The epithelial layer was formed by ciliated columnar epithelium in single rows or layers, transitional epithelium and stratified squamous epithelium. The stratified columnar epithelium predominated. There were no indications of keratinization. The stroma consisted of a delicate loose edematous connective tissue containing many blood vessels. The retiform spaces contained many plasma cells, lymphocytes, polymorphonuclear leukocytes and occasional Russell's bodies. The malignity of hard nasal papillomas is a disputed question. In this case numerous serial sections showed no signs of infiltration. Obstruction of nasal breathing is usually the only clinical symptom. Diagnosis requires repeated biopsies as differentiation from carcinoma is difficult. Cases with malignant degeneration have been observed. Recurrences are the rule, but two cases of permanent cure through roentgenization have been reported. The author recommends radical extirpation followed by roentgen irradiation.

EDITOR'S ABSTRACT.

TUMORS OF THE NASAL CAVITY. K. H. MICHEL, Frankfurt. Ztschr. f. Path. **43**:354, 1932.

The tumors of the nasal cavity and of the ethmoid bone and also those arising in the region of the palate, upper lip and orbits, as a rule, described as endotheliomas or cylindromas, are closely related to organoid tumors arising from the salivary glands. Histogenically, according to Mathias, they should be termed progonomas, because they might arise from misplaced tissue as the result of fetal atavism. All tumors of the maxilla which are suggestive of carcinoma should be examined carefully so as to rule out a possible origin in the parotid glands. This is of clinical importance because of the relatively lower malignancy of tumors arising in the parotid gland.

SAPHIR, Chicago. [ARCH. PATH.]

LIGATION OF INTERNAL MAXILLARY ARTERY IN UNCONTROLLABLE EPISTAXIS. J. LÖER, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **22**:437 (June) 1932.

Löer says that not infrequently hemorrhages from the posterior part of the nose, which is supplied by the internal maxillary artery, resist all therapeutic measures and endanger life. In several cases recently observed, he cured the epistaxis by ligation of the internal maxillary artery. All of the patients were middle-aged, adipose and of the apoplectic type. The spontaneous hemorrhage occurred following moderate exertion, probably from rupture of an arteriosclerotic vessel. No therapeutic measures could bring the bleeding to a standstill. The focus could not be definitely located because of the intensity of the hemorrhage. In all cases clinical and roentgenologic examination excluded the existence of a tumor. Ligation was undertaken by the maxillary sinus route as suggested by Seiffert. This limits the region to be eliminated to the focus of the hemorrhage. After removal of the mucosa, the lateral and posterior walls of the sinus are resected. The artery is isolated from the buccal adipose tissue on the posterior wall and ligated. No trophic disturbances of the nasal mucosa were observed following ligation of the artery.

EDITOR'S ABSTRACT.

NASAL OR ORAL OPERATION OF CENTRAL CYSTS. E. WIRTH, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **22**:475 (June) 1932.

Wirth thinks that the best surgical procedure for small cysts of the superior maxilla is enucleation with primary suture of the oral wound. For middle-sized cysts that are not in communication with the maxillary sinus, an individualized procedure is advisable. In the author's experience with cases of large cysts of the upper jaw, nasal methods of operation have proved effective. The objections made by dentists against the connection with the nose have generally not been found valid and cannot restrict the indications to the nasal methods. Protracted secretion from the nose was rarely noticed. The surgical connection of infected cysts with a healthy sinus had no bad results. Notable injuries to the teeth, or a tendency to caries or pulp necrosis were not observed even after complete removal of the cystic sac. With Denker's method, disturbances of sensitivity of the adjacent teeth were found more often than when this method is used for other conditions, but these appeared to have no drawbacks and did not inconvenience the patients. On the basis of forty-five operations on cysts of the teeth, the author thinks that Denker's operation, with complete removal of the cyst, is the method of choice in the treatment of large cysts of the upper jaw, without danger of recurrence or disturbance of oral and nasal function and with good cosmetic results.

EDITOR'S ABSTRACT.

Miscellaneous

RECENT ADVANCES IN PEDIATRIC OTOLARYNGOLOGY. LEE WALLACE DEAN, J. Pediat. **1**:379 (Sept.) 1932.

The clinical significance of the nasal sinuses in infancy is discussed. The differential diagnosis between an edematous lining of a nasal sinus caused by an allergic reaction and a thickened lining due to infection can almost always be made by a cytologic and bacteriologic study of the discharge coming from the sinus. Roentgen examination of the sinuses in infancy is important to show the presence and size of the sinuses, which vary greatly. The necessity of removing infected nasal and pharyngeal lymphoid tissue causing sinusitis and otitis is emphasized. Roentgen therapy may be used for the lymphoid apron occurring on the posterior pharyngeal wall after tonsillectomy. The peculiarities of the anatomy and the pathologic changes of the ear and mastoid in infancy are described. A study of the differential blood count and of the bacteriology of the aural discharge is of great importance in the course of infections of the ear.

LEMASTER, Evanston, Ill. [AM. J. DIS. CHILD.]

SUCCESSIVE BRONCHIAL FOREIGN BODIES. C. N. PEELER and V. K. HART, South. M. J. **25**:729 (July) 1932.

The authors report a case of successive bronchial foreign bodies aspirated in a girl aged 18 months. The bodies were aspirated in the same year, both were nonopaque and were in the right main bronchus, with obstructive emphysema on that side.

Prompt recovery followed the removal of each.

SCHLUTZ, Chicago. [AM. J. DIS. CHILD.]

A CASE OF FOREIGN BODY IN THE LARYNX OF A ONE YEAR OLD CHILD. A. CODINACH, Bol. Soc. cubana de pediat. **3**:371 (Oct.) 1931.

The author describes a case of a foreign body in the larynx in a 1 year old child. The patient presented a typical clinical history with the sudden onset, cyanosis, partial aphonia and fever.

The condition was not diagnosed until four months after the onset of the symptoms, i. e., at the time of the patient's admission to the hospital. The diagnosis was confirmed by roentgenologic and laryngoscopic examination. In spite of successful removal of the foreign body, bronchopneumonia developed, and the patient died.

The author brings out two points of interest in this case: (1) the long duration of symptoms, and (2) the unusual location of the foreign body (i. e., it was situated in the supraglottic region, firmly attached to the right vocal cord).

He mentions that in the series of Chevalier Jackson of 1,485 cases of foreign bodies removed from the upper respiratory passages there were only 5 similar cases.

SCHLUTZ, Chicago. [AM. J. DIS. CHILD.]

DEATH DUE TO LOCAL ANESTHESIA WITH PROCAINE HYDROCHLORIDE. T. SEEGER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **132**:49 (July 23) 1932.

In connection with the report of two cases in which death occurred immediately following the peritonsillar injection of a solution of procaine hydrochloride and epinephrine, Seeger discusses the literature on deaths caused by procaine hydrochloride. Analysis of the cases does not bear out the explanation of status thymicolymphaticus frequently given, generalized procaine hydrochloride poisoning or of other hypotheses advanced. In 53 per cent of the cases anesthesia was in the cervical region; in the majority of the other cases, it was in the lower part of the spinal column. The author discusses the clinical importance of the carotid sinus reflex, as shown by Hering and others, and the possibility of irritation of the sinus nerve presented by all the cervical cases reported. He thinks that the reported deaths following cervical procaine hydrochloride anesthesia did not result from procaine hydrochloride poisoning in the accepted sense, but from the unintentional production or exclusion of the carotid sinus reflex. The probable cause of death after sacral and lumbar anesthesia he assumes to be a rise in spinal fluid pressure. Death from procaine hydrochloride anesthesia that is technically well executed is to be feared only when the region to be anesthetized is one in which relatively insignificant mechanical injury may lead to severe disturbances and death. The rare manifestation of this latent danger might be accounted for by a special individual hypersensitivity in the region of certain reflex mechanisms or special anatomic characteristics of the anesthetized region such that structures not intended to be influenced become involved. In addition to the usual precautions of procaine hydrochloride anesthesia, attention should be given to the disposition of the patient and to the topography of zones where reflexes originate. Therapeutic measures against threatening manifestations should not be guided by the usually mistaken assumption of procaine hydrochloride poisoning.

EDITOR'S ABSTRACT.

Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY

H. P. Schenck, M.D., *Reporter*

Oct. 19, 1932

Ralph Butler, M.D., *Chairman*

The Nature of the Static Labyrinth as Determined by Experiments in Amphibia. Dr. William J. McNally, by invitation.

As has long been known, sensory hair cells, similar in general structure to those found in the cristae of the ampullary sacs, are found in the utriculus and sacculus. Each collection of hair cells, together with the supporting cells, is designated as a macula, the macula utriculi being found in the utriculus and the macula sacculi in the sacculus. Lying among the hairs of the hair cell are found masses of small crystals of calcium carbonate, the otoliths or otoconia. The utricle and saccule in man have two otoliths, but in animals there are frequently more.

The kinetic function arises from the semicircular canals, while the otoliths serve as a static mechanism. The usual conception of utricular function is that it is concerned with posture. These organs are assumed to give information regarding the position of the head when at rest and when making progressive—that is, nonrotatory—movements, supplementing, therefore, the functions of the semicircular canals on the supposition that the latter act especially in movements of rotation. According to this view, the otoliths act as a means of mechanical stimulation of the hairs. Being heavier than the endolymph, they press on the hairs with a force varying with the position of the head and thus give rise to sensations adapted to the maintenance of equilibrium. A sort of continuous discharge of such reflexes is assumed to occur from the contact of the otoliths with the hair cells in the dynamic organ of equilibration.

If both labyrinths are removed from a frog, it may assume normal or unusual positions, but it maintains its position in spite of the lack of labyrinths, and therefore the position is due to a neuromuscular mechanism. Change is the essence of stimulation. If the utricle is responding to a steady pressure, then it is the only known sensory epithelium that is so acting.

When a normal frog is placed on a plane and the plane is tipped slowly so that the movement is below the threshold of stimulation of the canals, the frog responds by discontinuous shifts. If the utricle were steadily stimulated, the shift would be steady and not discontinuous.

If one labyrinth is removed from a frog, the animal leans to one side. By manipulating such a frog and bringing its posture to the midline, the frog maintains its new symmetrical position when released. The moment the head is jarred or moved, however, the frog regains the asymmetrical position. A decerebrate frog is normal in labyrinthine movements, but voluntary movements are curtailed. It is the necessary jar of the utricular otolith that causes response. There is no steady pressure, but there is a sudden stimulus.

By removing all six canals in a frog, only the utricles remain as active vestibular end-organs. The posture is normal at rest, but any spontaneous movement causes the animal to sway and the jumps are not straight. If the utricles are removed as well, the swaying immediately disappears. The canal response is so rapid that response occurs in $\frac{1}{25,000}$ of a second.

In the frog, maintenance of position is evidently a muscular affair. When the canals are present, they act to keep the head level and prevent the sway which the utricles would otherwise bring about. The static mechanism is evidently muscular.

DISCUSSION

DR. B. H. SHUSTER: I do not know whether the conclusions are correct or not so far as this experiment is concerned, but this work in neuro-otology is of great importance. Dr. McNally's work has been brilliantly planned and directed with practical applications in mind. In showing that the anterior semicircular canal controls certain muscles, the neuromuscular mechanism may be histologically proved.

PULMONARY COMPLICATIONS OF TONSILLECTOMY WITH REPORT OF THREE CASES. DR. JAMES A. BABBITT.

Hemorrhage, anesthetic shock and pulmonary involvement are the three grave complications which may follow the operation for the removal of tonsils and adenoids. It would be natural to assume that pulmonary complications occurred through some carelessness in routine or some predisposing pathologic process. Yet this has happened after thirty years of active service in connection with operations on the tonsils in several hospitals, with scarcely a case of definite pulmonary complication as a sequence—three cases have occurred in services under my direction, two of lung abscesses and one of pneumonia, in the past two and one-half months, and these have followed what were reported as perfectly normal tonsillectomy and adenoidectomy.

Operations were done after routine reference from the outpatient department, with the customary preliminary safeguards performed in each case by a different operator, under ether anesthesia, with the patient in the prone position and with the use of the suction tube.

CASE 1.—C. M., a boy, aged 3, was admitted to the Children's Hospital on Aug. 8, 1932, for operation on the tonsils and adenoids; he was sent home next day. On August 11, he was brought to the medical dispensary with a temperature of 103 F. Four days later he returned to the nose and throat dispensary. The throat was about normal, but the left ear was injected. Four days later he returned again; the left tympanic membrane was incised, the right was noted as dull, and he was admitted to the hospital with a temperature of 100 F. Both tympanic membranes were incised on August 20, but the temperature continued to fluctuate between 100 and 103 F. Blood studies showed red cells just below 4,000,000 and white cells between 11,200 and 10,300 with from 70 to 72 per cent polymorphonuclears. A cough and foul breath developed. Roentgenograms of the mastoid and sinus were negative. Examination of the sputum showed mostly hemolytic streptococci. A roentgenogram of the chest revealed a large, dense area filling the upper lobe and a circular layer within this reaching from the second to the fourth rib, due either to abscess or to cavity. Medical consultation found bronchovesicular breathing with occasional râles in the right side of the chest anteriorly. Bronchoscopic drainage was begun, and cultures of the bronchial secretion showed predominence of hemolytic pneumococci with a moderate amount of nonhemolytic streptococci and other strains. Biweekly bronchoscopic treatment and postural drainage were given by Dr. Gabriel Tucker. The appearance of the mastoid was suspicious, but both ears have cleared and the child is practically well.

CASE 2.—E. D., a girl, aged 5, was operated at the Mary Drexel Hospital for hypertrophied and diseased tonsils on Sept. 9, 1932. Slight bleeding from the left tonsil required one suture. The day following operation the temperature rose to 102 F. This continued to be irregular for a week, from 100 to 101 F., then rose to 104 F. five days later. The crisis occurred the next day. The child coughed up much mucus, and the temperature gradually dropped to normal. The nose, throat and ears appeared normal the first week. On the ninth day fine râles were heard at the base of the left lung, and the next day diagnosis of bronchopneumonia was made. Roentgen examination showed marked infiltration at the

roots of both lungs and clouding of the left lower and lower half of the upper lobe of the lung. No cough or other subjective symptom was noted until the ninth day. Examination of the blood gave conventional results with the highest leukocyte count of 16,000. Examination of the sputum showed, among other flora, a few fusiform bacilli and spirilla. Final roentgenographic examination on September 30 showed thickened pleura and some infiltration at the root of the left lung but no consolidation or fluid. The child was discharged on September 30 in good condition.

CASE 3.—C. A., a girl, aged 3, with chronic nasal discharge and septic tonsils, was admitted to the Children's Hospital and the tonsils and adenoids were removed on Sept. 9, 1932. She was seen three days later in the nose and throat dispensary, and the throat was healing well. She was brought to the medical dispensary two days later with a temperature of 101 F. Two days later she was admitted to the hospital with a temperature of 100.8 F. Examination of the chest showed slight limitation of respiration and increased tactile fremitus over the upper lobe of the right lung and impaired resonance with coarse bronchial râles over both sides. For a few days the symptoms in the chest increased, with difficulty in coughing up mucus, but the temperature remained about 102 F. The first roentgenogram on September 15 showed an area of mottled density with poorly defined borders in the fifth interspace extending to the sixth interspace posteriorly on the right side. This was diagnosed as lobar pneumonia or beginning lung abscess. The diaphragm on the right side was elevated, but the heart and trachea were in normal position. A roentgenogram the next day showed widespread, finely mottled infiltration, scattered throughout the right lung and to a lesser extent in the left. This suggested tuberculosis. Four days later, infiltration was found scattered throughout both lungs—more marked in the right. The diagnosis was in doubt between tuberculosis, bronchopneumonia and multiple small abscesses. The child has been improving under treatment, and a later roentgenogram, October 4, showed diminution of the mottled area. The condition presented the initial flare, if not the actual pulmonary abscess, with a tuberculous background. Bronchoscopic drainage has been delayed.

DISCUSSION

DR. GABRIEL TUCKER: Only one important point concerns the bronchoscopist in these cases and that is the matter of treatment. It is imperative that diagnosis be made early and that bronchoscopic treatment be institued as soon as it is indicated. This type of therapy must be emphasized because it is the only effective means of combating the condition.

DR. GEORGE M. COATES: I think it is an excellent thing to bring these accidents to our attention and make us more careful. I should like to ask Dr. Babbitt to what he attributes these abscesses. I should also like to ask Dr. Tucker whether he as a bronchoscopist sees many of these cases following tonsillectomy. I suspect many of them are not being reported in the literature.

DR. BENJAMIN D. PARISH: Usually these cases occur once in two or three thousand cases, and here Dr. Babbitt runs into several in a short time. In the experimental production of lung abscesses in dogs, the condition appears to be the result of embolism plus infection. The jugular is tied off and and the clot infected. The septic clot is then injected into the opposite jugular, and, infection being present, lung abscess will result. Even after local anesthesia, the bronchoscopist can demonstrate some blood in the tracheal tract. I was unfortunate enough to see one of these cases some years ago; the patient died, refusing to go to the hospital.

DR. GEORGE WOOD: I have had one case in which the infection was inspiratory. The child got well with bronchoscopic treatment by Dr. Tucker. A small plug was coughed up a few days later followed by the discharge of pus. The clot was made up of clotted blood and milk. I experimented on dogs but could not produce lung abscesses, while rabbits developed abscesses if a mere shot was introduced into the lung.

DR. LOUIS H. CLERF: The aspiratory theory has the strongest evidence in support. In seventy-seven cases we found seventy-three operations had been performed under general anesthesia and four under local. Only 10 per cent occurred between infancy and 10 years of age. Lesions of the upper lobe were more common than of the lower lobe. The depth of anesthesia is probably the important thing.

DR. KARL M. HOUSER: I had one experience four or five years ago. A girl, 25 years of age, was operated on under local anesthesia. Two days after her discharge from the hospital, pain in the chest and a dry cough developed. The temperature was 104 F. The roentgenologist reported an abscess in the upper lobe of the left lung. There was no expectoration. Expectant treatment alone was used; the temperature dropped to normal, and at the end of two weeks the patient went home. At the end of three years a roentgenogram of the chest was negative. The pain and dry cough were probably due to an embolus which fortunately was not septic.

DR. WILLIAM MOORE: It cannot be definitely settled just how these complications occur. What interests me is what one can do to avoid them. My colleagues and I concluded that the most important thing in an adult was not to give morphine. The reflexes are then present at the time they are needed. In answer to our query the reply was invariably that morphine had been given. To operate with the head low helps somewhat, and after-care also helps.

DR. MATTHEW ERSNER: I am glad that Dr. Moore mentioned the fact that some form of opiate had effect. Were any of these patients given atropine preoperatively? Atropine produces dryness of the throat and bronchial tree. If atropine is given there is cessation of activity on the part of the ciliated epithelium. One must not administer anesthesia too deeply. Another serious question should be asked. Who operated on these patients? Go into any hospital, any afternoon, and you will find residents doing most of the tonsillectomies. Too often the operation is not done under light anesthesia and the operation takes too long. It is fortunate that more of these serious accidents do not occur.

DR. PHILIP STOUT: After the completion of the operation the patient is allowed to come out from under the influence of the ether alone. With the few abscesses that are seen, one may see some in patients whose tonsils are not removed. Some abscesses developed secondary to sinus disease. One physician had sinus trouble, and an abscess of the lung developed without any previous operation.

DR. GABRIEL TUCKER: I saw about twelve such cases last year. Infection occurs in the upper lobe because the patient is not in the upright position after operation and is in the recumbent position during anesthesia. Aspiration can thus occur in the upper as well as in the lower lobe.

DR. JAMES A. BABBITT: I had one experience with local anesthesia. The patient was a man of 70 years, who exhibited a rise in temperature after operation. A roentgenogram revealed collapse of an upper lobe. This collapse was due to mucus rather than the common cause. The child with pneumonia, whose case I reported, had no symptomatology, and the condition was not discovered until the crisis occurred. I agree with Dr. Ersner that in operations on the tonsils the operator should not be chosen promiscuously. A minute dose of atropine was given each of these patients before operation.

PRESENT-DAY THEORIES CONCERNING THE ETIOLOGY AND PATHOLOGY OF NASAL SINUS INFECTIONS. DR. BENJAMIN D. PARISH.

Textbooks during the past years have devoted many pages to operative work and treatment, but have been somewhat indefinite on etiology and pathology. Lately, however, the journals have been replete with reports delving into the etiology and pathology of sinus infection. For purposes of discussion, Dr. Parish adopted Eggston's classification of sinus disease. The first type considered, hyper-

trophic or polypoid sinusitis, is characterized by thickened and edematous changes in the mucous membrane and periosteum and is usually associated with polypoid masses of the soft tissues and rarefaction and osteoporoses of the bone. The second type, that known as atrophic, fibrotic, sclerotic or arteriosclerotic sinusitis, is characterized by an increase in the fibrous connective tissue, stroma, thinning of the mucous membrane and metaplasia of the surface epithelium, associated with thickening of the periosteum and a condensing osteitis. The third type is a combination of the first two types in the same patient.

The histopathology of these types was minutely described and attention then called to the striking similarity of these findings to those observed in a study of cases definitely diagnosed as allergic. The histopathology of allergic alterations was elaborated. In all of the work that has been done in the study of the regeneration of mucous membrane in these sinuses, all the investigators are struck by the fact that the regenerated epithelium is practically the same as the original tissue. A perfect pseudostratified ciliated columnar epithelium and complete regeneration of the epithelium and glands occurs in from three to five months. Regeneration, while extremely persistent, is slightly retarded in the presence of active infection. The significant point is that much of the pathologic process that was present before operation may readily recur in the newly formed tissue. This is especially true in allergic cases.

It is impossible to connect the etiology or course of the disease with the changes found. It is a question whether vascular changes are primary, whether polyps are a primary cause or whether the thickened basement membrane or even the round cell infiltration are where the pathologic process starts. For microscopic sections, reliance must be had on only advanced cases in which the process has gone far enough to justify the drastic step of operation. More information concerning the early changes is sadly needed. Many factors may well be the inciting cause of sinus infection. It is an accepted fact that something more than pure bacterial infection is required in the etiology of sinus disease. General bodily debilitation and lowered resistance from prolonged exposure, mental overwork, loss of sleep, types of diet, endocrine imbalance and anatomic factors, both acquired and hereditary, play a distinct part in the etiology. An important factor in the sinusitis of childhood has been said to be a lack of vitamin A. Diets rich in vitamin A and with increased vitamin B content were found of benefit. Lack of vitamin A has been shown to lead to atrophy in the turbinates of a rat.

Mechanical irritation, as the accidental introduction of water into the sinuses in swimming and diving, has led frequently to outbreaks of sinus infection. A rather important phase of the matter is the vasomotor factor in some of these infections, especially in the atrophic types; it has been felt that there was some pathologic process connected with the vasoconstrictors and vasodilators. The problem of allergy and allergic reaction in the nasal mucous membrane is one that deserves much attention and has much to support it. The mere fact that the pathologic process in the sinus is strikingly similar in a series of cases of allergic rhinitis and in a series of cases picked at random is significant. Possibly most of the cases of sinus disease have an allergic background. The persistent finding of eosinophils in the discharges and in the tissues is an argument for the allergic idea. It is still a question, however, which comes first, the allergy or the suppuration. Can one account for the fact that some patients showing other symptoms of allergy, asthma, eczema and similar conditions, in addition to the nasal condition, are completely cured by treatment and clearing up of the ethmoids, while others may be operated on, the ethmoids excised, etc., and yet neither the nasal symptoms nor the other allergic features subside? Dr. Parish feels that this may be accounted for by the fact that there are two types of suppurative ethmoiditis, that in which the allergic state is the background and the bacterial infection is merely secondary, and those cases in which the infection may be primary. Where infection is primary, radical treatment produces a cure. The greatest problem is to distinguish between the two types.

DISCUSSION

DR. CURTIS C. EVES: I feel that a great many of these cases are caused by an unhygienic diet. Especially in children, I feel that diet and hygienic regulations are more important than treatment. Modern life is responsible for much of this. The automobile, exposure to air, gas and tire dust and similar new conditions explain the increase in these cases. The modern craze for swimming is a factor, as the water washes off mucus and the mucous membranes are left unprotected. Swimming animals possess special mechanisms whereby the mucous membranes are protected Persons tend to blow the nose more vigorously when water gets into it. Controlling the habits and diet of patients with nasal sinus infections is most important.

DR. PHILIP STOUT: I have had some experience with these allergic cases. In nearly every case of asthma there is a pathologic process in the sinuses. Dark sinuses are found both by roentgenograms and by transillumination. Washing often removes little or nothing. Recurrence takes place a year or two after operation, and when the sinus is reopened it is found as infected as before the first operation. It is possible that the etiologic factor is a mold. Eosinophilia is said to be the opposite of suppuration.

DR. JAMES A. BABBITT: I am interested in one point. The ethmoidal sinuses were emphasized as a general point of infection in the sinus group. I have been impressed in the past by the large number of sphenoidal sinuses reported as normal. I wonder what Dr. Parish's experience has been?

DR. PARISH: I agree with Dr. Babbitt that the sphenoids are least often infected, unless the patient has been referred by an ophthalmologist. The roentgenologist most often reports the ethmoidal sinuses as showing involvement.

SOME STUDIES OF PATHOLOGIC TEMPORAL BONES IN CHILDREN. DR. ROBERT M. WOLFF.

The intention of the study was to supplement, from a macroscopic standpoint, the excellent work on microscopic sections of the ear both abroad, and more recently, in this country. An attempt was made to demonstrate a pathologic process in the middle ear and mastoid, and frequent sequelae of this process, viz., meningitis, brain abscess and lateral sinus thrombosis. A series of temporal bones was obtained in which there had been definite otitis media or mastoiditis, followed by intracranial complications. While symptomatology, diagnosis and treatment were not included for purposes of brevity, an effort was made to explain the occurrence and possible pathways of infection of these complications. The cases were followed, autopsies attended and temporal bones sectioned macroscopically to obtain the necessary data.

In order to appreciate fully what may happen in the course of a chronic or acute suppurative otitis media, a review of the anatomy of the infant and the adult was given. The possible pathways of infection and resulting pathologic process were discussed in detail, and the literature dealing with these was reviewed.

To obtain the temporal bones intact, including a small portion of the external auditory canal, without causing external deformity, a portable motor with a flexible shaft attached to a specially constructed circular saw was used. When a difficult angle was encountered, small drill holes were placed in line and the bone gently pried away from its attachments. The greatest possible precaution was taken to use the least amount of jarring in removing the bones. The specimens were immediately placed in Klotz solution no. 1 for twenty-four hours. They were then transferred to Klotz solution no. 2 in order to preserve as nearly as possible the original color of the mucous membrane and bone. After photographing the temporal bone, the specimens were frozen with dry ice and cut into sections approximately one-eighth inch in thickness. Several specimens were cut almost parallel with the external auditory canal. The remainder were cut at right angles to it. This was accomplished by the use of the saw and motor mentioned before. The saw blade being quite thin and with small teeth, there was only a fractional

amount of tissue lost in this method of cutting. Each section was examined carefully for evidence of bone necrosis, pus and adhesions and was photographed in series, first, from without inward; then, the reverse side. Owing to the thickness of these specimens, this double photographing was necessary since the small structures of the ear would otherwise have been missed. The sections were then mounted in flat glass jars, suspended so that both sides could be studied. The solution in the jars was again Klotz no. 2.

The first patient, a boy, 13½ months of age, was admitted to the hospital with a discharging right ear, a temperature of 103 F. and a leukocyte count of 22,000. A roentgenogram of the mastoid areas revealed cells of the semidiploic type and the right side definitely hazy as compared with the left. Clinically, the child did not appear to have mastoiditis. Two days after admission the aural discharge decreased and the drum was incised with release of pus. On the following day the spinal fluid contained 80 cells per cubic millimeter, but the fluid was sterile. A neurologic consultant felt that a brain abscess or localized cerebritis had resulted from mastoid infection. The leukocyte count was now 32,000 with 74 per cent polymorphonuclear cells, and weakness was noted on the left side of the face, left arm and left leg. A simple mastoidectomy revealed fairly normal bone and no frank pus. Cultures from the mastoid were reported sterile. The temperature continued to be irregular and high, and while the wound healed promptly, the ear continued to drain foul pus. Several blood transfusions and hypodermoclysis bolstered the patient's general condition, and for two weeks following operation the five specimens of spinal fluid obtained were reported sterile and of normal cell content. A furuncle in the right frontal region was discovered, and when opened a large amount of thick yellow pus was drained. A roentgenogram showed absorption of bone in this area. A week later generalized convulsions developed, the spinal fluid became cloudy with 6,800 white cells and 94 per cent polymorphonuclear cells, and hemolytic streptococci were found in cultures. Antistreptococcus serum was given intraspinally, but the child died one week later.

At autopsy no macroscopic connection between the middle ear or the mastoid and the brain cavity could be found. The dura was firmly adherent over the entire right side. There was an osteomyelitic area in the right frontal bone. A large subdural abscess, containing several drachms of pus, and enclosed by dense adhesions, was found in the right parieto-occipital area, and in the posterior part of the right frontal lobe, a small opening led to the osteomyelitic area in the frontal bone, the drainage route for the brain abscess. Definite meningitis extended over the entire left side of the brain.

This temporal bone was cut in a plane parallel to the posterior surface of the petrous portion. There was much pus in the middle ear and the same was true of the labyrinth. No definite areas could be found showing how the pus traveled from the ear to the brain. However, with a purulent labyrinthitis, there were several pathways available for the infection to extend into the brain cavity. This pathway was not macroscopically evident. The labyrinthitis was probably an extension of the middle ear infection through the round window, since this showed some bony necrosis around the margin. The specimen also shows well marked tympanic cellulae.

The second patient, a girl, aged 9 years, had had a discharging right ear for one month. On admission there was marked stiffness of the neck, overactive knee reflexes and a positive Kernig sign. The Babinski reaction was negative. Pus filled the right external canal and all drum landmarks were gone. The temperature was 103 F. Extensive and necrotic mastoid cells were revealed following a simple mastoidectomy. The lateral sinus was uncovered, but the sinus wall appeared healthy. Beneath the tegmen antri and extending deeply were some cells that were necrotic. These were followed to a depth where it seemed advisable to stop, due to the proximity of the superior semicircular canal. The spinal fluid from the first tap was cloudy and showed a cell count of 3,000 increasing later to 13,000. A hemolytic streptococcus was cultured from the spinal fluid and a pure culture of B. influenzae was obtained from the mastoid wound at operation. Eight days after admission the left arm and leg became flaccid. The mastoid wound showed

no tendency to heal and drainage was profuse. On the day of death, the respirations dropped to 6 per minute but later rose to 20. The heart continued to beat for ten minutes after respirations ceased.

At autopsy, the brain was markedly edematous, causing a blockage of the cerebrospinal fluid and an increased intracranial pressure. The surface of the brain was covered with a thick, plastic exudate, typical of a purulent, generalized meningitis of streptococcic origin. The dura over the petrous bone appeared normal and intact, until it was stripped off, when a dehiscence was revealed over the tegmen antri as well as necrotic bone just external to the internal auditory meatus. Slight pressure over the dehiscence above the mastoid excavation caused pus to be forced up through the necrotic bone. There was no evidence of an extradural collection of pus or walling off process. When the right temporal bone was removed, including the lateral sinus groove, the specimen was cut at right angles to the external auditory canal. A roentgenogram of the mastoid had shown diploic bone formation, but on sectioning there was evidence of pneumatic cell development of wide distribution. Cells were found above and below the bony wall of the internal ear as well as along the course of the eustachian tube. The bone in the extreme petrous tip showed some cell formation, perhaps accounting for the inward extension of infection rather than rupture through the external cortex. From the depths of the mastoid wound, the direct extension of the infection could be followed along the upper group of cells above the bony wall of the internal ear and just under the petrous cortex to become continuous with the dehiscence at the internal auditory meatus. B. influenzae may have accounted for the bone necrosis found deep in the temporal bone, as this organism has especial ability to attack bone. It is difficult to explain why with a pure culture of influenzal organisms in the mastoid a hemolytic streptococcic meningitis should develop. The B. influenzae in this case may have been one of the secondary invaders of the mastoid, and at the time of operation was the predominating organism, for one must concede the fact that the meningitis developed at least several days before admission and operation.

DISCUSSION

DR. GEORGE M. COATES: For a long time it has been known that at birth the middle ear is filled with embryonal tissue. In one of the cases the middle ear is shown still filled with embryonal tissue at 13½ months of age.

DR. PHILIP STOUT: I attend every autopsy I can. When an opening is made into the middle ear in infants and children, a jelly-like substance is found. It is occasionally found even in adults. Whether it occurs just before death or not, I do not know.

DR. WOLFF: It is difficult to explain the persistence of this material in the middle ear. In this case I assumed that the embryonal tissue had not yet disappeared.

Book Reviews

Oral Spirochetes and Related Organisms in Fuso-Spirochetal Disease. By David T. Smith, Associate Professor of Medicine, Duke University. Price, $4.50. Pp. 243. Baltimore: Williams & Wilkins Company, 1932.

This book presents an excellent, comprehensive work on the oral spirochetes and related organisms with the many diseases that the author believes are caused by them. David T. Smith has been a leading advocate of the fusospirochetal etiology of abscess of the lungs and bronchiectasis. His convincing investigations have led to an increasing acceptance of his views as opposed to those who consider the fusospirochetes as secondary invaders rather than as the primary pathogens.

In this book of 243 pages, well illustrated, he brings together the evidence for these organisms being the primary etiologic agents in noma, Ludwig's angina, hospital gangrene, tropical ulcer, balanitis, occasional infections of the ears and sinuses, cases of bloody bronchitis, most cases of abscess of the lungs and bronchiectasis and some cases of appendicitis, as well as being the now generally accepted cause of Vincent's angina, trench mouth and pyorrhea alveolaris. There are 822 references, every statement being supported by work the author himself has done or by reference to the published work of others.

The author summarizes his thesis in the introduction: "While there is no evidence that oral spirochetes alone can produce disease in man, they are probably the most important member of a symbiotic group of anaërobic organisms capable of initiating a severe and often fatal disease. This group, composed of spirochetes, fusiform bacilli, vibrios and cocci is surpassed only by syphilis and tuberculosis in the wide variety of lesions it produces in the body. Situated in the tonsils and about the teeth they are in a strategic position to initiate disease locally in the tonsils, gums, cheeks, tongue, pharynx and larynx, or to invade the eyes, ears or sinuses. They may be aspirated and infect the trachea, bronchi or lungs, swallowed and cause disease in the esophagus, appendix or colon, or by emboli reach the brain, lungs or other internal organs.

"The chief characteristics of the lesions produced are necrosis, pseudo-membrane formation on the surface and fetid odor. The diagnosis is easily established by staining a smear or by the darkfield apparatus. The treatment is open drainage and exposure to the air, the application of oxidizing agents, and arsenic therapy."

Directory of Otolaryngologic Societies *

INTERNATIONAL

Second International Otolaryngological Congress

President: Prof. Dr. A. G. Tapia, Hermosilla, 2, Mod, Madrid, Spain.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde, Heereweg 143, Gronigen, Holland.

FOREIGN

New Zealand British Medical Association, Ear, Nose and Throat Section

Chairman: Dr. James Hardie Neil, Auckland.
Secretary: Dr. W. Gillies Borrie, Auckland.

Royal Society of Medicine, Section of Laryngology

President: Dr. E. Musgrave Woodman, 22, Newhall St., Birmingham, England.
Secretary: Dr. A. Lowndes Yates, 33 Weymouth St., W. 1, London.
Place: London. Time: May 5, 1933.

Royal Society of Medicine, Section of Otology

President: Dr. J. F. O'Malley, 6, Upper Wimpole St., W. 1, London.
Secretary: Dr. C. Gill Carey, 106, Harley St., W. 1, London.

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Harris P. Mosher, 828 Beacon St., Boston.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: Milwaukee. Time: June 12-16, 1933.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
President-Elect: Dr. J. M. Wheeler, 30 W. 59th St., New York.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Boston. Time, 1933.

American Bronchoscopic Society

President: Dr. Richmond McKinney, 899 Madison Ave., Memphis, Tenn.
Secretary: Dr. Edwin McGinnis, 104 S. Michigan Ave., Chicago.

American Laryngological Association

President: Dr. George Fetterolf, 2010 Spruce St., Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Place: Washington, D. C. Time: May 9-10, 1933.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

American Laryngological, Rhinological and Otological Society

President: Dr. Joseph C. Beck, 185 N. Wabash Ave., Chicago.
Secretary: Dr. R. L. Loughran, 33 E. Sixty-Third St., New York.
Place: Chicago. Time: June 8-10, 1933.

American Otological Society

President: Dr. George L. Tobey, Jr., 270 Commonwealth Ave., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. Fortieth St., New York.
Place: Washington, D. C. Time: May 9-11, 1933.

SECTIONAL

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. R. H. Seeley, 9 Court St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

New England Otological and Laryngological Society

President: Dr. Henry A. Barnes, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Lyman G. Richards, 319 Longwood Ave., Boston.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Hans Barkan, 490 Post St., San Francisco.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: San Francisco. Time: June 28-30, 1933.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. John H. Harter, 1215 Fourth Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. R. B. Smith, Alma, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. John B. Gregg, 101 S. Main Ave., Sioux Falls, S. D.
Secretary-Treasurer: Dr. Frederick H. Roost, 420 Trimble Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Wiley R. Buffington, 211 Camp St., New Orleans.
Secretary: Dr. W. D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.

Southwestern Michigan Triological Society

President: Dr. W. E. McGarvey, 802 Jackson City Bank Bldg., Jackson.
Secretary: Don C. Lyon, 1405 Union and Peoples National Bank Bldg., Jackson.
Time: Third Thursday of alternate months.

STATE

Colorado Oto-Laryngological Society

President: Dr. Harry L. Whitaker, 1612 Fremont St., Denver.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. M. H. Gill, 36 Pearl St., Hartford.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Thomas H. Hall, Jr., 617 Mulberry St., Macon.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. Robert J. Masters, 805 Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. J. Kent Leasure, 611 Hume-Mansur Bldg., Indianapolis.
Place: Indianapolis. Time: Dec. 13, 1933.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. W. Pearson, 410 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. W. F. Boiler, 105 East Iowa Ave., Iowa City.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Wilfred Haughey, 303 Post Bldg., Battle Creek.
Secretary: Dr. H. O. Westervelt, 239 Pipestone St., Benton Harbor.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Harold J. Rothschild, 239 Lowry Bldg., St. Paul.
Secretary-Treasurer: Dr. W. E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month, October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. W. J. Marshall, 212 N. Higgins Ave., Missoula.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Time: Semi-annually.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Charles H. Schlichter, 556 N. Broad St., Elizabeth.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.
Secretary: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Axel Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Walter Bristow, Medical Arts Bldg., Columbia.
Secretary: Dr. J. W. Jervey, Jr., 101 Church St., Greenville.
Place: Columbia. Time: October, 1933.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. D. Jones, 3116 Live Oak St., Dallas.
Secretary: Dr. A. F. Clark, 1034 Nix Bldg., San Antonio.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Erza Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Frank P. Smart, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. Henry Grant Preston, Professional Bldg., Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.
Secretary: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Elbert A. Curtis, 65 Central St., Newark.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. L. C. Rouglin, 127 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore City Medical Society, Section on Otology and Laryngology

President: Dr. James J. Chisolm, Medical Arts Bldg., Baltimore.
Secretary: Dr. Louis Diener, 2449 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

Buffalo Oto-Laryngological Society

President: Dr. John F. Healey, 925 Delaware Ave., Buffalo.
Secretary-Treasurer: Dr. Herrmann E. Bozer, 468 Delaware Ave., Buffalo.
Place: Buffalo Club. Time: Second Tuesday of each month from September to May, inclusive.

Chicago Laryngological and Otological Society

President: Dr. Charles Younger, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

Cincinnati Oto-Laryngological Society

President: Dr. Henry M. Goodyear, The Doctors' Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. M. W. Jacoby, 2323 Prospect Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 1065 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

College of Physicians of Philadelphia, Section on Otolaryngology

Chairman: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Clerk: Dr. Karl M. Houser, 1826 Pine St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. John B. Alcorn, 167 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Edward W. Harris, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. O. M. Marchman, 1717 Pacific Ave., Dallas, Texas.
Recording Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Corresponding Secretary: Dr. J. M. Potts, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. C. F. Howland, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Ia.
Time: Third Monday of each month, September to May, inclusive.

Detroit Oto-Laryngological Society

President: Dr. R. Lee Laird, 513 David Whitney Bldg., Detroit.
Secretary-Treasurer: Dr. J. M. Sutherland, 8-257 General Motors Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Secretary: Dr. Van D. Rathgeber, 1011 Medical Arts Bldg., Fort Worth, Texas.
Place: University Club. Time: 6.30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 26 Sheldon Ave., Grand Rapids, Mich.
Place: Various local hospitals. Time: 6:30 p. m., third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. J. F. Gamble, 201 Main St., Houston, Tex.
Secretary: Dr. B. F. Payne, 1300 Walker Ave., Houston, Tex.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. Albert N. Lemoine, 906 Grand Ave., Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Ferris L. Arnold, 923 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Clyde E. Harner, 314 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

Acting President: Dr. D. E. Godwin, 910 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Mary Louise Cafe. Time: 6:30 p. m., fourth Monday of each month, September to May, inclusive.

Medical Society of the County of Kings, Section on Otolaryngology

Chairman: Dr. Edward Leo Berger, 903 Park Pl., Brooklyn.
Secretary-Treasurer: Dr. Oscar Rodin, 893 Eastern Parkway, Brooklyn.
Place: Library Bldg., County Society, 1313 Bedford St., Brooklyn.
Time: 9 p. m., second Wednesday of January, March, May and November.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. Albert P. Tibbets, 1801 I St., Washington.
Secretary: Dr. G. Victor Simpson, 1710 Rhode Island Ave., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. T. F. McCormick, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville.
Secretary-Treasurer: Dr. H. C. Smith, Medical Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month, October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. Charles A. Bahn, 150 Baronne St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

New York Academy of Medicine, Section of Otolaryngology

Chairman: Dr. Charles Imperatori, 108 E. 38th St., New York.
Secretary: Dr. David H. Jones, 140 E. 54th St., New York.
Place: 103d St. and 5th Ave. Time: 8:30 p. m., third Wednesday of each month from October to May.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. L. G. Howard, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: Dinner, 6 p. m. Program, 7 p. m., third Wednesday of each month from October to May.

Philadelphia Laryngological Society

President: Dr. Louis H. Clerf, 128 S. Tenth St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

Pittsburgh Otological Society

Chairman: Dr. Roy C. Cooper, 510 S. Aiken Ave., Pittsburgh.
Secretary-Treasurer: Dr. Alexander R. Hampsey, 806 May Bldg., Pittsburgh.

Richmond Ophthalmological and Oto-laryngological Society

President: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. E. S. Amsler, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. William E. Sauer, 3720 Washington Blvd., St. Louis.
Secretary: Dr. B. J. McMahon, 813 Missouri Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Charles J. Boehs, 1126 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert Carson Martin, 384 Post St., San Francisco.
Secretary: Dr. George N. Hosford, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. J. L. Scales, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 505 Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. David Hartin, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. W. W. Henderson, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. G. Dickinson, 109 S. Warren St., Syracuse, N. Y.
Secretary-Treasurer: Dr. Harold H. Joy, 109 S. Warren St., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy W. Dunlap, 108 W. 6th St., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, 108 W. 6th St., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.